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The Journal of

Thoracic and Cardiovascular Surgery

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29, 126

JANUARY
VOL. 39, ~~DECEMBER~~-JUNE, 1960
THE C. V. MOSBY COMPANY 1960 ST. LOUIS

VOLUME 39

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The Journal of
Thoracic and
Cardiovascular Surgery

VOLUME 39
JANUARY 1960
NUMBER 1

Original Communications

THE RELATIONSHIP OF POSTOPERATIVE ACIDOSIS TO PULMONARY AND CARDIOVASCULAR FUNCTION

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THE NORMAL response to trauma which leads to recovery is becoming established.[29] Made up, as it is, by the intricate combination of compensatory functions of several organ systems, many variations are possible. Yet, only through knowledge of the general pattern and the degree of variation in a given patient can an estimate of his postoperative state be achieved. Following thoracic surgical operations, the system which most obviously fails is the circulation. Yet, in the majority of instances, this is secondary to respiratory inadequacy which results in chemical changes of the blood, detrimental to cardiovascular function.

From observations made upon patients subjected to pulmonary resection and other endothoracic procedures, it is the purpose of this paper to outline the pattern of the circulatory and the respiratory response during recovery in the first week. A comparison of their normal relationship with the situation existing in several patients who were hypotensive at various times during the recovery period suggests that, in man, prolonged acidosis associated with hypoxia leads, if not corrected, to sudden death from circulatory collapse.

Beecher and Murphy[2] drew attention to the profound acidosis which may develop during thoracic operations. Since that time, considerable work has been

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This work was supported in part by a grant from the National Institutes of Health, HC 1317-C-6.

Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

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done on the mechanism of this carbon dioxide retention,[42] its effects on the circulation[7, 37] and the nervous system.[8] Gibbon and associates[18] and subsequently Martin and Stead[28] and Hood and Beall[23] noted the depressed respiration and the hypercapnea present in the immediate postoperative period. Stead[41] and Björk[4] made observations on the respiratory function later in the postoperative period. Yet, little exact information is available on the behavior of the circulation in relation to the blood chemical changes occasioned by such alterations in respiration as may take place. With this in mind, 45 patients who underwent thoracic surgical procedures were studied. Of these, 5 died subsequent to the operation and 3 others had transient episodes of hypotension relieved by correction of respiratory inadequacy. This permitted a comparison with the remainder who made uncomplicated recoveries. Blood gas and electrolyte studies were related to pulmonary function and hemodynamic observations during and after the operation. In 14 of the patients, by employing dye dilution curves, data were obtained on the cardiac output to suggest what the usual circulatory picture may be as a result of thoracic surgery.

PROCEDURE AND METHODS

Each patient selected for study of the plasma electrolyte pattern and the circulatory and respiratory responses occasioned by the performance of a thoracotomy underwent a schedule of tests. These consisted of measurements of cardiac output and other hemodynamic studies, determinations of pulmonary function, and analysis of the blood gas and certain of the plasma electrolytes. As far as practical, these were performed simultaneously and generally in the following sequence: (1) several days preoperatively, (2) following induction of anesthesia and intubation of the trachea, (3) near the completion of the endothoracic procedure, (4) early in the postoperative period (3 to 5 hours after closure of the chest), (5) on the morning of the first day postoperatively, (6) the second day, (7) the third or fourth day, and (8) after 1 week.

This was accomplished at the time of the respiratory studies by placing a Cournand needle in a peripheral artery. Through this, blood was drawn for analysis as well as for the performance of dye dilution curves. The dye was injected through a polyethylene catheter inserted through a thin-walled No. 14 needle in the antecubital vein to the point where its tip was estimated to be in the superior vena cava. On the morning of the operation, the venous catheter was placed and kept open by a very slow drip of 5 per cent glucose in water. It was used as the site for intravenous injection of anesthetic agents as well as the infusion of blood during the procedure. It was left in place for 1 or 2 days postoperatively, and was used during this time for such intravenous fluid therapy as was necessary, as well as for the measurement of cardiac output.

A random selection was made of 45 patients from the Thoracic Surgical Service of the Cleveland Metropolitan General Hospital (formerly Cleveland City Hospital) for this study during the past 3 years. No effort was made to secure "bad risks" or for that matter to avoid them. Patients who underwent cardiac operations were not included in this group because of their known

peculiarities in fluid and electrolyte balance[31] which add another set of problems to the study of the circulatory, respiratory, and electrolyte changes caused by thoracotomy.

These patients were divided into three sections: (1) those undergoing pneumonectomy, (2) partial resection (lobectomies, segmental, and wedge resections), and (3) thoracotomy for operations upon structures other than the lung. Of the 5 patients who died, all in the postoperative period from 4 to 10 days later, 4 had undergone pneumonectomy and 1 a lobectomy.

The 10 patients who had pneumonectomies ranged in age from 37 to 64, with an average of 49 years. Five were operated upon for tuberculosis and 5 for cancer. The average maximum breathing capacity was 75 ± 12 per cent of the predicted normal and, in only two instances, was there an elevation of the walking ventilation above 11.5 L. per square meter surface area per minute.

Of the 25 patients upon whom a partial resection was performed, 16 had tuberculosis, 1 cancer, and the others a variety of infections. The average maximum breathing capacity was 78 per cent of the predicted normal. The maximum breathing capacity of 3 only was below 55 per cent. One of these subsequently died.

Ten patients underwent intrathoracic operations other than upon the lung. Of these there were two esophagectomies, three repairs of hiatus hernia, three decortications, one closure of bronchopleural fistula and thoracoplasty, and one exploration without resection. The patients' ages ranged from 40 to 66, with an average of 55. With the exception of 2 of the patients subjected to decortication, who had maximum breathing capacities below 55 per cent and vital capacities below 53 per cent of normal, the respiratory function of this group was fair, with an average maximum breathing capacity of 78 ± 10 per cent of normal.

None of the patients was hypertensive. In 3, there was evidence of previous myocardial damage or arrhythmia by electrocardiogram, or walking ventilation values above 11 L. per square meter per minute in 3. One of these subsequently suffered auricular fibrillation following lobectomy.

In all the patients reported, a combination of Pentothal (for induction only), nitrous oxide, ether, and oxygen was employed for anesthesia. In 14, Anectine was used at the time of intubation of the trachea. The majority were ventilated during thoracotomy by manual compression of the bag. A mechanical respirator was employed during all or part of the procedure in 7. Postoperative care included the use of oxygen by nasal catheter for 1 day, or more, if needed. Fluids were limited to blood replacement and approximately 1,500 c.c. of 5 per cent glucose in water during the first 24 hours. Thereafter, the patients, with the exception of those who had esophagectomy, took fluids by mouth.

All of the patients underwent study in the pulmonary function laboratory by such tests as were deemed necessary to arrive at a decision concerning the suitability and risk of the proposed operation. This included maximum breathing capacity, walking ventilation, timed vital capacity, function fluoroscopy, and bronchospirometry.

Thereafter, in the subsequent periods outlined, the respiratory observations made were on respiratory rate, tidal volume, and vital capacity. These were done with the aid of a Collins recording respirometer.* The preoperative control tests of this group were made with the patient reclining in bed under circumstances similar to those encountered postoperatively. No ventilatory measurements were made during anesthesia. Although tidal volume and respiratory rate were measured within the period of 3 to 5 hours after the operation, it was often impossible to obtain meaningful records of vital capacity due to the patient's inability to cooperate.

The hemodynamic observations included arterial blood pressure and pulse rate in all patients. The first was obtained by auscultation with a sphygmomanometer on an arm. Venous pressure determinations were made serially in 10 patients by the free fall of saline in a spinal manometer attached to the vena caval catheter. The zero line was placed at the estimated level of the right auricle.

Serial cardiac output determinations were made in 14 of the patients. The principle of measurement of cardiac output by the use of dye dilution curves recorded from the arterial system depends upon the injection of a measured amount of dye into the venous circulation as close to the heart as possible.[21] It is diluted by the blood pumped through the heart, and flow can be calculated by dividing the quantity injected by the average concentration of the dye measured in the arterial blood during the period of one circulation. The quantity of dye in the continuously sampled arterial blood can be measured photometrically,[33] and produces characteristic curves of concentration during the first and second circulation. The area of the first curve extrapolated at its termination to the base line can be converted to milligrams of dye per liter of blood by comparison with calibrations from known dilutions of the dye in samples of the patient's blood.

The dye used for injection was Indocyanine Green.†[16] Arterial blood was drawn through a densitometer (Model XC-100A‡) and the electrical output recorded on a high impedance recorder amplifier (Model 126-127§). Constant flow of arterial blood through the densitometer at 7.64 c.c. per minute was obtained with an infusion-withdrawal pump (Model 600-900‖). The quantity of dye injected ranged from 0.07 to 0.14 mg. per kilogram of body weight. A planimeter was used to calculate the area under the curve with the descending limb extrapolated to the base line. Calibration of deflection of the curve was carried out after each set of determinations by drawing heparinized samples of the patient's blood containing known dye concentrations through the densitometer at the standard rate of 7.64 c.c. per minute. From the arterial blood pressure determinations obtained with a sphygmomanometer at the time of the dye curve, mean pressure was approximated as the average of systolic and

*Warren E. Collins Company, Boston, Mass.
†Hynson, Westcott, and Dunning, Baltimore, Md.
‡The Waters Corporation, Rochester, Minn.
§The Sanborn Company, Cambridge, Mass.
‖Harvard Apparatus Co., Inc., Dover, Mass.

diastolic. Peripheral resistance was calculated in absolute units[19] by the formula:

$$\text{Total Peripheral Resistance (dyne seconds/cm.}^{-5}) = \frac{\text{Mean Arterial B.P. (mm. Hg)} \times 1{,}332}{\text{Cardiac Output cm.}^3\text{/sec.}}$$

Arterial blood gas determinations were made upon samples obtained at each period of observation. Drawn into oiled syringes sealed with mercury, the pH was measured with the Cambridge pH meter* and corrected for temperature. These results were frequently checked in a pH meter heated to 38° C. which showed little deviation from the calculated results.†

The per cent of oxygen saturation of the blood was calculated from the nomogram of Dill,[11] employing pH and pO_2 values. The latter was determined in approximately one half of the specimens by the bubble technique of Riley and his co-workers.[34] For the remainder, oxygen content, as measured by the method of Van Slyke and Neill,[43] was compared with capacity to arrive at a value for the per cent of saturation. When both were available, they were checked against each other. In general, there was close agreement.

The carbon dioxide tension was calculated employing the nomogram of Singer and Hastings[39] which requires knowledge of the hematocrit, pH, and carbon dioxide content obtained from Van Slyke analysis.[43] More than one half of these values was checked again with close agreement by carbon dioxide tensions measured by the bubble technique.[34] In 11 instances where there was a discrepancy, the values obtained by the latter method were accepted.

Blood electrolyte values were obtained from the heparinized arterial samples taken at the times the respiratory and other tests were carried out. Sodium and potassium concentrations were measured with the Baird‡ flame photometer.

For the determination of ionized calcium, plasma from arterial blood collected under oil was subjected to ultrafiltration in a modification of the Lavietes cell.[26] This is so arranged that the filtrate is separated from the cellophane membrane by a layer of mercury to avoid the interference of dialysis. Both total and filtrable calcium concentrations were determined by the EDTA titration method of Carr and Frank.[6] In order to arrive at a value for ionized calcium from the nomogram of McLean and Hastings for the dissociation of calcium citrate,[22] it is necessary to know the concentration of citric acid in the filtrate which was obtained by the technique of Ettinger, Goldbaum, and Smith.[12]

Plasma lactate values were measured by the colorimetric method of Barker and Summersen.[1] Inorganic phosphorus and protein levels in the plasma were obtained by the methods, respectively, of Fiske and Subbarow[13] and Weichselbaum.[44]

RESULTS

Data concerning the circulation, respiration, arterial blood gas and pH, and the blood electrolyte pattern of the patients who survived thoracotomy are

*Cambridge Instrument Company, Cambridge, Mass.
†Courtesy Dr. Byron Bloor.
‡The Baird Company, Cambridge, Mass.

TABLE I. CIRCULATORY VALUES AND STANDARD DEVIATIONS OF SURVIVING PATIENTS

NUMBER	OPERATION	PREOP.	ANESTHESIA	OPERATION	POSTOPERATIVE PERIOD				
					3-5 HR.	1 DAY	2 DAYS	3-4 DAYS	1 WEEK
	Pneumonectomy								
7	Pulse rate	85 ± 7	95 ± 10	102 ± 10	94 ± 18	94 ± 20	91 ± 15	94 ± 12	92 ± 8
7	Arterial blood pressure	121 ± 12 / 72 6	116 ± 18 / 80 10	102 ± 24 / 60 10	104 ± 12 / 67 8	117 ± 10 / 78 7	112 ± 10 / 70 6	117 ± 12 / 70 7	120 ± 10 / 73 5
2	Cardiac output (% diff. from preop.)	4.6 ± 1.0 L./min.	−10 ± 8%	−32 ± 12%		+8 ± 5%	+22 ± 14%		+24 ± 11%
	Partial Resection								
23	Pulse rate	84 ± 8	100 ± 15	99 ± 10	101 ± 16	103 ± 18	111 ± 16	103 ± 10	95 ± 8
23	Arterial blood pressure	118 ± 14 / 74 7	122 ± 12 / 82 10	112 ± 26 / 68 12	109 ± 22 / 70 10	121 ± 16 / 73 5	118 ± 12 / 75 6	120 ± 10 / 75 5	121 ± 10 / 77 5
9	Cardiac output (% diff. from preop.)	5.9 ± 0.8 L./min.	−11.2 ± 13%	−33 ± 10%	+7.1 ± 4.1%	+9.3 ± 8.5%	+32 ± 28%		+16 ± 12%
	Other Thoracotomies								
10	Pulse rate	89 ± 5	100 ± 5	100 ± 7	100 ± 15	95 ± 14	97 ± 15	96 ± 11	93 ± 12
10	Arterial blood pressure	125 ± 12 / 78 4	115 ± 11 / 78 3	110 ± 26 / 65 8	120 ± 23 / 74 11	125 ± 20 / 81 3	126 ± 18 / 80 5	125 ± 14 / 78 4	125 ± 14 / 79 5
4	Cardiac output (% diff. from preop.)	5.2 ± 1.1 L./min.	−9 ± 15%	−30 ± 13%	+57 ± 29%	+45 ± 29%	+47 ± 40%		+21 ± 15%

given with their standard deviations in Tables I, II, III, and IV. To permit comparison of the effects produced at different stages of recovery by the three types of operative procedure, each table is divided to present separately the data from pneumonectomies, partial resections, and other intrathoracic operations. As may be seen from examination of the standard deviations, little significant difference exists in the observations obtained from the three groups. Attention will be drawn to those situations in which important variations do occur. For convenience, then, the average values for surviving patients are given in Figs. 1, 2, 3, and 4.

Five patients died postoperatively at times ranging from 4 to 10 days postoperatively. In each instance, this followed respiratory insufficiency from pneumonia. The data from these patients are presented in Table V. The course of a patient typical of those in this group who died is given in Fig. 5.

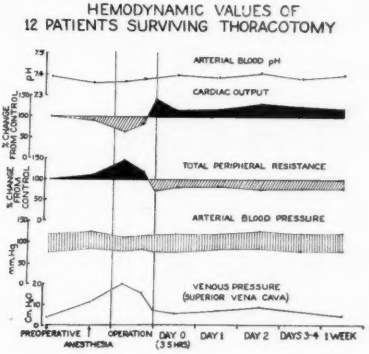

Fig. 1.

Circulatory Observations.—Little difference in blood pressure or pulse rate (Table I) existed in the response of the three surviving groups to the operation. Adequate data from serial observations are available on the cardiac output of 14 patients. The average preoperative cardiac output by the dye dilution technique was 5.33 L. per minute with a standard deviation of ±0.96 liter.

Although there was only a moderate and statistically insignificant depression of blood pressure during the induction of anesthesia and the operation, the average cardiac output fell 9 ± 5.9 per cent in the first period, and later in the operation to –32 ± 10.5 per cent from the control level. The latter is significant ($p < 0.02$). At this time, the average total peripheral resistance rose to 45 ± 13.4 per cent above the preoperative value of 1,857 dynes cm./sec.$^{-5}$ Later in the operation the cardiac output returned toward normal, being 16.2 ± 2.56 per cent below the preoperative level. The blood pressure was also returned to nearly normal values giving a calculated peripheral resistance 16 ± 9.8 per cent above normal.

The vena caval pressure promptly rose from 5.8 ± 1.5 cm. H_2O during the induction of anesthesia and intubation to 14.5 ± 6.2 cm. Throughout the re-

TABLE II. VENTILATION AND STANDARD DEVIATIONS OF SURVIVING PATIENTS

NUMBER	OPERATION	PREOP.	ANESTHESIA	OPERATION	POSTOPERATIVE PERIOD				
					3-5 HR.	1 DAY	2 DAYS	3-4 DAYS	1 WEEK
	Pneumonectomy								
5	Tidal volume (L./min.)	0.48 ± .10 (7)			0.40 ± .06	0.45 ± .07	0.47 ± .08	0.50 ± .10	0.52 ± .09
7	Respiratory rate	22 ± 2			25 ± 4	24 ± 4	21 ± 3	25 ± 4	22 ± 4
5	Vital capacity (L./min.)	2.81 ± .34 (7)				1.32 ± .16	1.34 ± .19	1.43 ± .24	1.65 ± .22
	(% normal)	69% ± 9% (7)				32%	34%	37%	41%
	Partial Resection								
8	Tidal volume (L./min.)	0.45 ± .07 (23)			0.34 ± .08	0.37 ± .06	0.40 ± .07	0.47 ± .06	0.52 ± .08
23	Respiratory rate	20 ± 2			26 ± 4	26 ± 5	26 ± 4	25 ± 4	22 ± 5
8	Vital capacity (L./min.)	3.12 ± .72 (23)				1.09 ± .31	1.12 ± .27	1.35 ± .25	1.35 ± .26
	(% normal)	81% ± 13%				28%	29%	36%	36%
	Other Thoracotomies								
4	Tidal volume (L./min.)	0.46 ± .13 (10)			0.40 ± .10	0.41 ± .09	0.39 ± .07	0.38 ± .07	0.45 ± .11
10	Respiratory rate	19 ± 3			27 ± 3	25 ± 5	26 ± 4	24 ± 4	23 ± 4
4	Vital capacity (L./min.)	2.78 ± .68 (10)				0.98 ± .28	1.13 ± .26	1.24 ± .38	1.44 ± .36
	(% normal)	73% ± 12% (10)				27%	30%	33%	38%

mainder of the operation it remained elevated between 15 and 18 cm. H_2O on the average, with a greatest standard deviation of ±4.82. Following extubation it returned to near preoperative levels where it remained through the convalescent period.

At the termination of the operation, when the tube was removed from the larynx, a slight fall in blood pressure took place in all patients to an average of 105/64 mm. Hg; but cardiac output increased in all. The average value was +40.2 ± 36 per cent above the preoperative level. The large standard deviation is due to the great rise in 2 patients to +210 and +232 per cent. At this time, peripheral resistance was found to be decreased 26 ± 7.7 per cent under the resting value. Up to 1 week, cardiac output remained elevated, the greatest being on the second postoperative day when it was 31 ± 23.9 per cent over the preoperative level. By the end of a week it had returned to 19.6 ± 16 per cent.

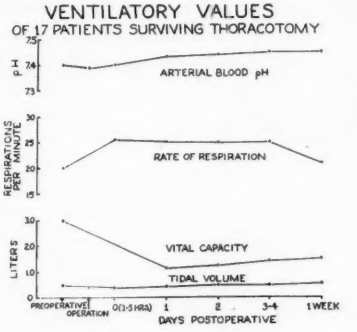

Fig. 2.

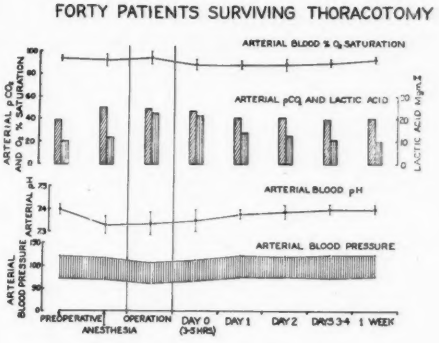

Fig. 3.

The blood pressure and pulse rate of patients who died postoperatively behaved in most respects in a fashion similar to those of the survivors, until 2 to 3 days prior to death. As shown in Table V, the pulse rate was significantly elevated above that of the survivors at a corresponding time. More important, the mean of the blood pressures was somewhat lower 2 days before death and significantly lower on the day before (94/52 ± 8/12 mm. Hg) and on the day of death (85/48 ± 7/20 mm. Hg). It was notable that blood pressure tended to change abruptly upward and downward during the last 2 days, as measured from hour to hour. In general, the pulse rate varied inversely as the blood pressure in this period. No consecutive data are available concerning cardiac output in this group.

Ventilation.—The patients who survived pneumonectomy, partial resection of lung, and other intrathoracic procedures were comparable preoperatively and showed no significant differences of respiratory rate in their tidal volumes, which averaged 460 ± 90 c.c., or the vital capacities which averaged 76 ± 12 per cent of the predicted normal. As shown in Fig. 2, there was a moderate reduction in tidal volume with an increase of respiratory rate in the early postoperative course. During the first day after the operation, the mean tidal volume of

TABLE III. BLOOD GAS VALUES AND STANDARD DEVIATIONS OF SURVIVING PATIENTS

NUMBER	OPERATION	PREOP.	ANES-THESIA	OPERATION	POSTOPERATIVE PERIOD				
					3-5 HR.	1 DAY	2 DAYS	3-4 DAYS	1 WEEK
7	*Pneumonectomy*								
	pCO_2	38 ± 4	54 ± 16	49 ± 10	41 ± 6	42 ± 8	41 ± 4	38 ± 4	40 ± 4
	pH	7.38 ± .02	7.32 ± .04	7.34 ± .06	7.40 ± .03	7.41 ± .02	7.41 ± .03	7.39 ± .03	7.40 ± .02
	% O_2 saturation	94 ± 4	93 ± 9	94 ± 4	93 ± 2	91 ± 5	92 ± 3	91 ± 3	93 ± 2
23	*Partial Resection*								
	pCO_2	40 ± 4	49 ± 7	51 ± 7	48 ± 3	42 ± 3	42 ± 2	41 ± 2	38 ± 2
	pH	7.40 ± .02	7.33 ± .05	7.32 ± .05	7.33 ± .05	7.39 ± .03	7.40 ± .01	7.40 ± .02	7.41 ± .01
	% O_2 saturation	93 ± 4	90 ± 6	95 ± 5	89 ± 5	86 ± 5	84 ± 6	89 ± 3	94 ± 3
10	*Other Thoracotomies*								
	pCO_2	38 ± 6	47 ± 4	48 ± 7	46 ± 4	39 ± 3	38 ± 2	36 ± 4	38 ± 2
	pH	7.40 ± .03	7.34 ± .04	7.35 ± .04	7.35 ± .02	7.38 ± .02	7.39 ± .03	7.42 ± .02	7.40 ± .02
	% O_2 saturation	94 ± 2	96 ± 2	97 ± 2	87 ± 6	88 ± 4	89 ± 6	91 ± 3	92 ± 5

those who had lobectomies (370 ± 60 c.c.) remained below that of the other two groups. The pneumonectomies averaged 450 ± 70 c.c. In the majority, tidal volume had returned to normal by the fourth day and in all by the end of a week.

Vital capacity on the day after the operation fell significantly in all cases to levels averaging 28 per cent of the predicted normal. This reduced the average respiratory reserve in the three groups to between 24 and 37 per cent of what it had been before the operation. As may be seen from Table II, this reduction of vital capacity was least among the postoperative pneumonectomies. By the end of 1 week, the average vital capacity reached a level of 51 per cent of the preoperative value or 38 per cent of normal. The patients who died did not show any statistically significant difference in respiration from the survivors, although tidal volume tended to be lower.

Arterial Blood Gas and Hydrogen Ion Concentration.—Preoperatively there were 5 patients with arterial blood oxygen saturations below 90 per cent. These ranged from 78 to 89 per cent. Of these, 1 had a moderately elevated pCO_2. Otherwise carbon dioxide tension was normal in all of the patients.

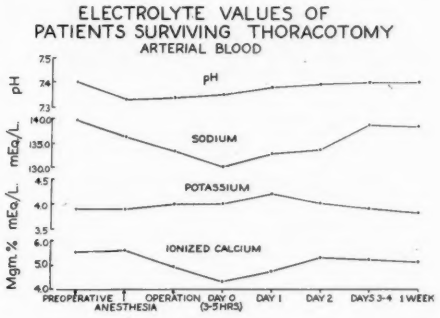

Fig. 4.

During the induction of anesthesia, 19 patients were found to be acidotic. Ten of these were grossly so, with arterial pH values below 7.30. Subsequently, in the course of the thoracotomy, acidosis was present in 21, 5 of them having pH values between 7.2 and 7.3. The pH of 3 was below 7.2. Three to 5 hours following the operation, acidosis was observed in 12 patients, although 5 of these had not been acidotic during the operation.

In each case, the acidosis was related to CO_2 retention. Carbon dioxide tension on the average, as shown in Table III, was above or at the upper limits of normal, with large standard deviations indicating the wide range of observations during these periods.

One case in particular is reported in this connection.

E. R. (U-83957), a 47-year-old Negro man, was admitted to Lowman Pavilion with a diagnosis of cavitary tuberculosis of the right upper lobe, 5 months prior to operation. He was treated with isoniazid and para-aminosalicylic acid. The vital capacity was 61 per

cent and maximum breathing capacity 78 per cent of predicted normal. Blood gas studies disclosed the arterial pH to be 7.40; pCO_2, 44; and oxygen saturation, 93 per cent. Blood chemical determinations were also within normal limits. The patient was in good condition and considered a satisfactory candidate for right upper lobectomy. This was undertaken on Sept. 11, 1958, and the operation was accomplished without difficulty in approximately 3 hours and 0 minutes. However, it was noted that the patient breathed at times with great effort. The blood pressure dropped from the preoperative level of 120/80 to 85/60 mm. Hg during induction and subsequently returned to 125/90 from which it gradually fell to 102/82 by the end of the procedure. As the chest was being closed, it was reported that the arterial pCO_2 in the blood sample, drawn an hour before, was 145 mm. Hg and the pH 6.91 while oxygen saturation was 93 per cent. Investigation disclosed that through an oversight, the CO_2 absorbing cannister had not been placed in the anesthesia machine. By slowly diluting the bag mixture with oxygen to avoid a sudden shift to pure oxygen, the pCO_2 was reduced. Within an hour, the arterial carbon dioxide tension was 62 mm. Hg while the pH was brought up to 7.21. The blood pressure fell to 72/50 mm. Hg. A gram of calcium chloride

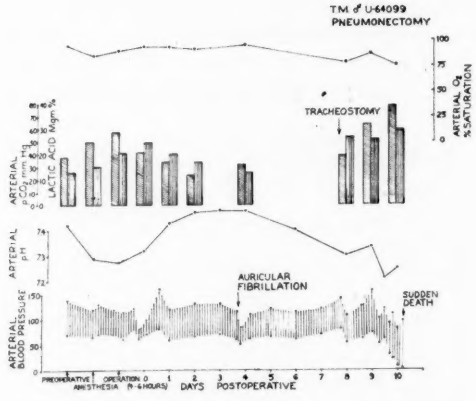

Fig. 5.

administered intravenously promptly restored it to 105/74 mm. Hg. During the subsequent 4 hours, the arterial pCO_2 was reduced to 36 mm. Hg, although the pH could not be made to rise, above 7.22. The blood lactic acid was 52 mg. per cent. The potassium rose to 5.9 mEq./L. while the ionized calcium dropped to 0.7 mM./L. At this time the patient was anuric, but urinary output started 4 hours postoperatively and quickly increased in volume. Within the next 3 hours, 750 c.c. was excreted. Concurrently with the onset of diuresis, the arterial pH rose slowly to 7.38. Except for some lassitude and a headache for 2 days, the patient made an otherwise uneventful recovery.

Throughout the postoperative period, the oxygen content of the arterial blood was maintained near normal in all but 2 of the patients surviving pneumonectomy. In these, it was depressed only for a single day during a critical period. On the other hand, as shown in Table III, there was a significant fall of hemoglobin oxygen saturation among the patients who underwent lobectomy. This was most noticeable on the second day postoperatively, when the average oxygen saturation had fallen to 84 ± 6 per cent from 93 ± 4 per cent ($p < 0.05$). This trend was less marked in the third group of patients who underwent operations on structure other than the lung, but was particularly noticeable among

the 3 patients who underwent decortication. On the second postoperative day, the hemoglobins were 81, 84, and 85 per cent saturated from preoperative control levels which ranged from 87 to 93 per cent saturated.

The data in Table V suggest that the blood gas values of the patients who died were not significantly different from the survivors during the operation and for 2 days thereafter. But when the available data for the 2 days preceding death are examined, it will be seen that, despite efforts to improve respiration by aspiration, tracheostomy, assisted ventilation, and other measures, there was a progressive and significant fall of oxygen saturation to an average of 76 ± 8 per cent on the day preceding death. The arterial pH also fell to 7.26. In 3 patients, values between 7.12 and 7.28 were recorded just before death. The pCO_2 rose reaching a level above 60 mm. Hg in the 3 patients from whom blood was drawn on the day prior to death. An example of this is shown in Fig. 5.

Blood Electrolytes, Lactate, and Phosphate.—Among the surviving patients there is little difference in the pattern of blood electrolyte response. A significant fall in sodium took place which was most pronounced on the afternoon of the operation and the following day. It was not unusual to find the plasma sodium below 128 mEq./L.

TABLE IV. BLOOD CHEMISTRY AND STANDARD DEVIATIONS OF SURVIVING PATIENTS

NUMBER	OPERATION	PREOP.	ANES-THESIA	OPERA-TION	POST-OPERATIVE PERIOD				
					3-5 HR.	1 DAY	2 DAYS	3-4 DAYS	1 WEEK
	Pneumonectomy								
5	Sodium (mEq./L.)	137 ± 6	134 ± 6	132 ± 5	135 ± 5	131 ± 3	133 ± 5	134 ± 4	137 ± 5
7	Potassium (mEq./L.)	3.9 ± .4	3.9 ± .4	4.0 ± .3	4.0 ± .4	3.7 ± .3	3.7 ± .3	3.9 ± .5	3.8 ± .4
6	Ion. calcium (mg. %)	5.8 ± .6	6.5 ± .5	5.0 ± .5	4.6 ± .4	5.0 ± .5	5.2 ± .4	5.3 ± .4	5.2 ± .4
6	Lactate (mg. %)	13 ± 4	16 ± 5	27 ± 5	17 ± 6	20 ± 4	13 ± 5	14 ± 5	10 ± 4
	Partial Resection								
20	Sodium (mEq./L.)	141 ± 6	139 ± 7	133 ± 4	131 ± 5	135 ± 6	135 ± 5	140 ± 7	139 ± 4
23	Potassium (mEq./L.)	3.9 ± .3	4.1 ± .5	3.9 ± .4	3.9 ± .5	4.4 ± .3	4.1 ± .3	3.9 ± .3	3.9 ± .3
23	Ion. calcium (mg. %)	5.3 ± .4	5.5 ± .2	4.9 ± .6	4.1 ± .4	4.5 ± .3	5.3 ± .3	5.2 ± .3	5.1 ± .4
23	Lactate (mg. %)	10 ± 4	17 ± 6	25 ± 7	26 ± 5	17 ± 6	16 ± 5	11 ± 5	9 ± 4
	Other Thoracotomies								
9	Sodium (mEq./L.)	138 ± 7	132 ± 5	135 ± 7	131 ± 6	129 ± 6	131 ± 7	138 ± 8	138 ± 6
9	Potassium (mEq./L.)	3.8 ± .3	3.6 ± .2	4.0 ± .3	4.1 ± .2	4.0 ± .2	4.0 ± .2	4.0 ± .3	3.7 ± .4
8	Ion. calcium (mg. %)	6.0 ± .5	5.0 ± .3	4.6 ± .3	4.6 ± .2	4.9 ± .3	5.3 ± .4	5.6 ± .3	
8	Lactate (mg. %)	11 ± 3	14 ± 5	21 ± 6	24 ± 3	19 ± 3	13 ± 3	14 ± 4	10 ± 3

The potassium levels were either not changed or only very slightly elevated on the first and second days postoperatively. These differences were not statistically significant.

The ionized calcium levels were depressed consistently and significantly ($p > 0.02$) in all groups on the afternoon of operation to a level 21 per cent below that found preoperatively. In general, these had returned to their control values by the second day.

The average concentration preoperatively of inorganic phosphate was 3.3 ± 0.5 mg. per cent which rose to a peak during the operation and in the immediate postoperative period to 4.5 ± 0.6 mg. per cent. This returned in the course of the next 2 days to the preoperative value. The blood lactate followed a similar pattern starting at 10.7 ± 4 mg. per cent and rising on the average to 24.4 ± 6. This ordinarily required 3 days to drop to normal. In those patients who were hypoxic or hypotensive, lactate values as high as 57 mg. per cent were seen.

Summaries of the histories of surviving patients who developed transient postoperative pulmonary insufficiency and hypotension follow.

W. B. (U-80326), a 62-year-old white man who underwent an uneventful pneumonectomy for tuberculosis on April 19, 1956, had a preoperative vital capacity of 69 per cent and a maximum breathing capacity of 80 per cent. In other respects also he was considered a satisfactory candidate for the operation. On the morning after the operation, he continued to be hypotensive (B.P. 80/58). Both physical examination and x-ray disclosed atelectasis of the remaining lower lobe. The arterial pH which had been normal before and during the operation was 7.29. The carbon dioxide tension had risen to 65 mm. Hg and the oxygen saturation was 82 per cent. Lactic acid and potassium were both moderately elevated, the latter to 5.8 mEq./L. Bronchoscopy was performed with the removal of a mucus plug giving good aeration of the lobe. Blood pressure promptly returned to 130/92 mm. Hg. An arterial blood sample drawn a few hours later revealed that pH, pCO_2, and oxygen saturation had become normal again. He made an uneventful convalescence thereafter.

T. C. (U-45764), a 48-year-old white man, was admitted for cavitary tuberculosis involving the right upper and middle lobes. The preoperative arterial pH was 7.36 with a pCO_2 of 40 mm. Hg and 92 per cent oxygen saturation. Vital capacity was 81 per cent and the maximum breathing capacity 69 per cent of normal. On April 30, 1957, he underwent a right upper and middle lobectomy and made a satisfactory recovery that afternoon. The next morning his blood pressure was found to be 78/52 and he appeared dyspneic and cyanotic, despite the administration of oxygen by nasal catheter. The arterial pCO_2 was 67 mm. Hg and the pH 7.28. The hemoglobin was 79 per cent saturated. Blood lactate had risen to 29 mg. per cent. Calcium and potassium levels were normal. Bronchoscopy was performed to relieve a left lower lobe atelectasis. The blood pressure rose to 120/68 mm. Hg with a decrease of pulse rate from 130 to 116. A blood sample the next day gave a pH of 7.38 and blood gas values within normal limits. The lactate had fallen to 18 mg. per cent.

A. L. (U-63478), a 42-year-old white woman, was admitted 13 months preoperatively for pulmonary tuberculosis. Preoperative pulmonary function studies disclosed that the vital capacity was 57 per cent and the maximum breathing capacity was 64 per cent of normal. The left lung was performing 28 per cent of the ventilation and 21 per cent of the oxygen uptake. Blood gas and electrolyte values were normal. On Jan. 9, 1957, she underwent a left pneumonectomy with essentially an uneventful convalescence until the fifth day when it was discovered that she had developed pneumonia in the right lung. On the sixth day, despite efforts at coughing and aspiration, many ronchi were heard. The blood pressure gradually fell from 110/72 to 92/64 mm. Hg with fluctuations up and down. A blood sample disclosed the arterial pH to be 7.30 whereas the pCO_2 was 67 mm. Hg. The blood was 82 per cent oxygen saturated. A tracheostomy was performed for suction and assisted respiration. Within 6 hours, the blood pressure had risen to 135/85. The next day the arterial pH was 7.37 and the pCO_2 44 mm. Hg. Oxygen saturation had risen to 89 per cent. The electrolytes were all normal except for the sodium which was 129 mEq./L. She subsequently made an uneventful recovery.

Two case summaries of patients who died postoperatively are presented here as typical examples of the type of respiratory problems which were followed by death.

C. V. (U-33193), a 57-year-old man, who entered for his fourth admission because of severe dyspnea of 6 months' duration. He had arrested tuberculosis and had developed emphysematous cysts of both bases, most marked in the right lower lobe. This seriously compressed the remaining lung tissue. The arterial blood had a pH of 7.41 with a pCO_2 of 52 mm. Hg. The hemoglobin was 78 per cent saturated with oxygen. Blood pressure was 110/68 mm. Hg. Despite a vital capacity of 40 per cent and maximum breathing capacity of 35 per cent, a right lower lobectomy and tracheostomy were performed on Feb. 12, 1958, in an attempt to improve the respiratory capacity. Postoperatively, he developed paralytic ileus and required gastric suction. The arterial oxygen saturation did not change, but the pCO_2 rose to 79 mm. Hg with an accompanying depression of pH to 7.20 on the afternoon of the operation. Pulse was near 110 and blood pressure 100/76. The next day, oxygen saturation was 69 per cent but pCO_2 had fallen to 52 mm. Hg, with a pH of 7.37. On the [illegible] appeared to be lethargic although the nail beds were pink. Bronchial breathing [illegible] the right upper chest, and x-ray studies suggested bronchopneumonia despite [illegible] of the trachea. At that time, the arterial pH was 7.32 and urine output had decreased to 22 c.c. per hour. The plasma sodium had fallen to 129 from 148 mEq./L. The potassium had risen to 5.9 mEq./L. The blood pressure varied between 120/90 and 90/50 mm. Hg. Temperature was 40° C. per rectum. On the morning of the fourth day, the situation was essentially unchanged, despite the use of assisted respiration through the tracheostomy with the Bennett valve. At this time, the pH of the arterial blood was 7.22 and the pCO_2 73 mm. Hg. The oxygen saturation was 68 per cent. His blood pressure remained near 90/68 mm. Hg, with a pulse rate of 120. Shortly thereafter he vomited, became cyanotic, and suddenly died.

T. M. (U-64099) (see Fig. 5), a 54-year-old Negro man, was admitted because of a cough of 1 month's duration. X-rays showed atelectasis of the left upper lobe. The vital capacity was 92 per cent and maximum breathing capacity 84 per cent of normal. Preoperative blood studies, including pH, pCO_2, oxygen saturation, potassium, and sodium, were normal. A diagnosis of pulmonary carcinoma was made, and a pneumonectomy was performed on Aug. 6, 1958. His postoperative course is illustrated in Fig. 5. Except for continued intrapleural bleeding postoperatively, requiring four additional blood transfusions, the course was uncomplicated until the seventh postoperative day when respiration became difficult due to secretions. Auricular fibrillation occurred and the rate was controlled with digitalis. A tracheostomy was performed with some improvement. However, his downward course continued. He died on the tenth day with a sudden fall of blood pressure. At autopsy a cerebellopontine metastasis was found.

DISCUSSION

The response of the circulation to thoracic surgical operations as observed in 14 surviving patients is shown in Fig. 1. The number of observations is too small to permit comparison in the various types of operations. Yet, the circulatory alterations fell into the same general pattern. The anesthetic and operative procedure produced a depression of cardiac output (-35 ± 5.2 per cent) with increased total peripheral resistance ($+45 \pm 13.5$ per cent). Postoperatively this was followed by an immediate rise of output, acutely stimulated at the time of extubation. This was accompanied by a decrease of resistance (-26 ± 7.8 per cent). Throughout the remainder of the recovery period, the

situation was unchanged. On the average, the cardiac output was at its greatest (+31 ± 13.9 per cent) on the second postoperative day. At that time, the peripheral resistance was –25 ± 3 per cent of the preoperative level. Toward the end of the week, both tended to approach normal values.

The observed decrease in cardiac output and the rise of central venous pressure during anesthesia are well known and have been shown to be independent of either acidosis or hypoxia.[14, 35] Depression of the myocardium by the anesthetic agent or an increase in pulmonary vascular resistance has been suggested as a cause.[25] The further depression of cardiac output during the operation to values as low as 51 per cent of the resting output may well have been the result of blood loss, yet it is probable that this also was related to a change in cardiac function. Calculation of stroke volume shows that following the induction of anesthesia it fell from an average preoperative level of 59 ± 8 c.c. to 52 ± 4 c.c. Later in the operation, the average stroke volume was 34 ± 4 c.c. which was a significant change. Possibly this is related to the fall of pH which has been so commonly found during such procedures.[2] Jerusalem and Starling[24] observed that the cardiac dilation caused by carbon dioxide retention "affected the systolic more than the diastolic volume, so that the ventricular output is diminished."

It is likely that the marked rise in cardiac output, observed following anesthesia as the patients were extubated, was the result of a stimulus resulting in a release of epinephrine. This is suggested by the marked rise of pulse rate. Possibly it is also provoked by increased circulatory demands during coughing and struggling.

During the days succeeding the operation, the increase in circulation was met by a rise of pulse rate which averaged 117 ± 5 in these patients rather than an increase of stroke volume. The latter (65 ± 7 c.c.) remained near the preoperative value of 59 ± 8 c.c. This observation of augmented output in the recovery period differs from that of Snyder[40] who found a fall of 41 per cent in 8 patients following abdominal operations. In his experience, 5 days were required for return to normal. It may be argued that the greater output at this time was the result of mild hypoxia which is a well-established cause for epinephrine release.[5] Gellhorn's[17] observations on man subjected to mild hypoxia suggested a rise of cardiac output. In this connection it is of interest that the least tachycardia was observed in the patients who had had pneumonectomies. They also showed the least hemoglobin oxygen desaturation. Furthermore, Stead[41] observed an increase of the metabolic rate 1 day following lobectomy and thoracoplasty amounting to 128 per cent of the preoperative value. This might well be expected to result in elevation of the circulatory flow.

Both Martin and Stead[28] and Hood and Beall[23] found that a large proportion of patients were underventilating within the first hour after the completion of thoracic operations, with a tendency toward a fall in arterial pH. At the time when measurements were made on this series of patients (3 to 5 hours after surgery), the tidal volumes were only slightly depressed and the pH values were within the normal range in almost all of the patients. The exceptions to

this were among those who had had lobectomies and who showed a significant depression of tidal volume with a marked increase in respiration. However, by employing Fowler's factor for normal anatomic dead space,[15] it appears that alveolar ventilation was only slightly depressed in the great proportion of patients, the average being –16 ± 12 per cent below the preoperative resting level among the survivors.

On the following day, the data in Table II indicate that tidal volumes were not significantly below normal and the respiratory rates were moderately elevated. Although subject to some error, by the means used to calculate them, the average of alveolar ventilation was only slightly depressed (–10 ± 12 per cent), not significantly so. This is in contrast to Stead's observation which showed a depression of 35 per cent.[41] However, his patients had been subjected to a combined pulmonary resection and thoracoplasty, making respiration more difficult. Fig. 2 shows how these nearly normal tidal volumes were maintained despite a marked reduction in the vital capacity and respiratory reserve.

It is impressive how exactly the pH and pCO_2 of the blood were maintained within the narrow margins considered to be normal from the first postoperative day onward in the surviving 40 patients. The only exceptions were the 3 patients whose histories are presented and who developed problems of ventilation and inability to keep the arterial pCO_2 below 48 mm. Hg for relatively short periods of time.

Although pCO_2 was maintained normally, there was a moderate oxygen desaturation in the arterial blood of almost all the patients. The data in Table III suggest that this was significantly greater among those who underwent lobectomy and may be related to edema caused by trauma to the lung left in situ. This phenomenon was also noted to a lesser extent in those patients who underwent operations upon other structures within the chest. Ordinarily, this was most pronounced on the second day when a hazy ground-glass appearance of the lung on the side of operation was seen by x-ray. Usually resolution took place within 4 days and was accompanied by a return toward normal of the hemoglobin oxygen saturation. Maier[27] noted this and suggested that blood stasis in the lung might be a factor in producing postoperative pulmonary complications.

The blood electrolyte changes in the patients who recovered are those usually observed following surgical trauma of this magnitude elsewhere in the body.[30] The depression of the sodium, greatest during the first 2 days after the operation, is a dilutional phenomenon caused by the retention of water and the lysis of cells and fat. It has been suggested also that sodium may enter the cells as a result of such tissue acidosis as may exist.[31] The rise of potassium is related to cellular damage and hypoxia.[36] Sealy and associates[37] and others[7] have demonstrated a transient rise of potassium when hypercapnia is reduced. Probably this played a part in the moderate hyperkalemia seen in 16 of the patients shortly after the operation. In none of them was found a level over 5.9 mEq./L., which is not sufficiently high to seriously affect the heart.

TABLE V. SUMMARY OF DATA FROM PATIENTS WHO DIED POSTOPERATIVELY

NUMBER					POSTOPERATIVE PERIOD					
								DAYS PRIOR TO DEATH		
		PREOP.	ANESTHESIA	OPERATION	4-6 HR.	1 DAY	2 DAYS	2 DAYS	1 DAY	DAY 0
	Circulatory									
5	Pulse rate	90 ± 12	95 ± 11	122 ± 16	103 ± 12	118 ± 7	110 ± 12	120 ± 8	124 ± 13	128 ± 8
5	Arterial blood pressure	135 ± 8 / 70 5	140 ± 30 / 70 20	108 ± 12 / 60 6	99 ± 5 / 70 5	112 ± 12 / 70 5	110 ± 12 / 70 6	106 ± 15 / 64 9	94 ± 8 / 52 12	85 ± 7 / 48 20
	Ventilation									
2	Tidal volume (L./min.)	0.55 ± .04(5)				0.34 ± .03	0.36 ± .03	0.40 ± .04	0.55 ± .10	
5	Respiratory rate	24 ± 3			30 ± 4	29 ± 2	30 ± 4	32 ± 3	34 ± 6	36 ± 5
2	Vital capacity (L./min.)	2.29 ± 1.0(5)				1.18 ± .41	1.32 ± .34			
	(% Normal)	56%				28%	32%			
	Blood Gas									
4	pCO_2	42 ± 4	51 ± 8	60 ± 10	55 ± 5	53 ± 4	48 ± 4	54 ± 4(3)	64 ± 6(3)	81 ± 15(3)
5	pH	7.41 ± .03	7.36 ± .04	7.29 ± .07	7.29 ± .08	7.34 ± .04	7.36 ± .06	7.32 ± .02(3)	7.26 ± .06	7.21 ± .06(3)
4	% O_2 saturation	92 ± 4	82 ± 6	72 ± 14	86 ± 6	89 ± 4	85 ± 10	83 ± 8(3)	76 ± 8(3)	73 ± 5(2)
	Electrolytes									
4	Sodium (mEq./L.)	139 ± 6	134 ± 5	135 ± 3		135 ± 4	129 ± 4		133 ± 3(3)	
5	Potassium (mEq./L.)	4.3 ± .2	4.6 ± .4	4.8 ± .8	5.0 ± .6	5.1 ± .4	4.8 ± .3		4.9 ± .3(3)	5.5 ± .3(2)
4	Ion. calcium (mg. %)	5.0 ± .5	4.8 ± .6	4.6 ± .6	4.0 ± .3	4.3 ± .3	4.7 ± .5	4.8 ± .2	5.1 ± .3(2)	
5	Lactate (mg. %)	11 ± 4	17 ± 3	20 ± 5	23 ± 5	16 ± 3	14 ± 4			34 ± 4(2)

Note: Figures in parentheses indicate number of determinations if different from the numbers at left.

The depression of ionized calcium in the blood has been reported before[9] and is probably the result of retention of inorganic phosphate and the renal loss of calcium following citrate infusions.[45] In no instance was a level below 0.75 mM./L. seen. This also is above the level known to result in cardiovascular failure.

Metabolic acidosis, or better "hypoxic acidosis," took place to some extent in all of the patients during the operation, probably due to inadequate perfusion of part of the vascular bed while cardiac output was decreased. This required 2 to 3 days to return to normal levels. However, the lactate was seldom more than moderately elevated on the morning after operation. This suggests that at this time the majority of the tissues were being maintained with adequate perfusion and reasonably normal oxygen tensions.

Three patients who ultimately survived and 5 others who died postoperatively manifested serious cardiovascular depression or failure. Examination of the case histories and the data in Table V discloses that this circulatory difficulty was preceded by severe respiratory abnormalities. This has been noted before and various explanations offered. From the observations here reported, it appears that the common factors resulting from the respiratory inadequacy were a prolonged period of progressively increasing acidosis and a moderate to severe hypoxia.

Stead[41] suggests that exhaustion might be responsible for the failure to maintain the blood gasses at normal levels. Björk[3] states that a decrease of ventilated alveoli decreases diffusion surface necessitating greater ventilation and produces unfavorable mechanical factors with decreased lung compliance. It is true that the patients in this series were breathing at levels near their respiratory reserve, but 3 of the 4 who died did so after assisted respiration had been started. Since all showed hemoglobin oxygen saturation ranges from 68 to 82 per cent while breathing oxygen, it appears that a decrease of the ventilation-perfusion ratio[10] had taken place. This produced a state of hypoxia and metabolic acidosis for which the remaining lung was unable to compensate by the elimination of CO_2. The arterial carbon dioxide tension was over 65 mm. Hg and the pH below 7.30 in all 7 patients. Those who died had been moderately acidotic for at least 2 days before death. An examination of the blood electrolyte levels during the hypotensive period failed to disclose any abnormalities sufficient to account for the circulatory failure.

Intact animals made to breathe high concentrations of CO_2 show serious hypotension only at onset and termination of the hypercapnia.[7, 37] That this is true in man is suggested by the observations here cited on the patient whose arterial pCO_2 was 145 mm. Hg for at least 90 minutes during his operation. As was pointed out, a number of patients were quite acidotic immediately following the operation without serious sequelae, if the pH was normal on the following morning. Thus, it appears that short periods of intense respiratory acidosis are not lethal.

Nahas and Cavert[32] have pointed out that failure is rapidly induced in the isolated heart by acidosis if circulating epinephrine is not present. It might be

argued that certain of these patients lacked normal adrenal function. In none of the 3 upon whom autopsies were performed was adrenal disease found. Furthermore, all of the patients showed a normal tachycardia postoperatively and often exhibited fast pulses until just prior to circulatory failure and death. Large swings of pulse and blood pressure were exhibited on the day before death in 3 of the patients. This is similar to the pattern in anoxic dogs observed by Clowes, Hopkins, and Simeone[7] prior to cardiac arrest. These findings all suggest that the sympathetic and adrenal systems were functioning in these people.

The fact that the acidosis was prolonged and accompanied by various degrees of hypoxia may be the important matter. Hypoxia of this degree is compatible with life. Gremels and Starling[20] noted little change in the heart's capability of performing work until oxygen saturation fell to 75 per cent, and failure did not occur until a critical level of 25 per cent oxygen saturation of the blood was reached.

This situation differs from that encountered in emphysematous patients who are gradually adapted to a combination of hypercapnia and often quite severe hypoxia. Thus, it appears that the presence of acidosis may limit the ability to compensate for even moderate degrees of hypoxia in the relatively acute state.

From a therapeutic standpoint, active measures to prevent or correct prolonged acidosis should be undertaken when possible. The maintenance of normal oxygen tensions may be important in avoiding the serious circulatory consequences of acidosis. Therefore, the administration of oxygen is indicated whenever acidosis is suspected postoperatively except for special circumstances as in patients with emphysema.[38] Finally, arterial punctures and the determination of the pH at times may be of great assistance in the direction of the postoperative care of patients who have undergone thoracic operations.

SUMMARY

1. The cardiovascular response to the trauma of thoracic surgery and to the shifts of blood gas and electrolyte values occasioned by alterations of the respiratory function was studied in 45 patients. The operative procedures included pulmonary resections and others, but none upon the heart. Observations were made before, during, and for a week after the thoracotomy.

2. In general, blood pressure was maintained near normal with only transient depression in the immediate postoperative period. However, during the operation, cardiac output fell 33 per cent on the average, with a decrease of stroke volume. This was accompanied by a rise of total peripheral resistance. Postoperatively this situation was promptly reversed, when the cardiac output rose with a return of normal stroke volume.

3. Although vital capacity and respiratory reserve were depressed to less than one half of those found preoperatively, tidal volume and alveolar ventilation remained near normal except for a few hours after the operation.

4. All of the patients showed moderate postoperative oxygen desaturation of the arterial blood, the least being among those who had been subjected to

pneumonectomy. However, the pH and pCO_2 were maintained within the narrow limits considered normal except for the operative period and for a short time thereafter.

5. The blood sodium fell on the average to 130 mEq./L. on the afternoon of the operation returning to normal by the third or fourth day. The potassium rose slightly while the ionized calcium level fell 21 per cent to the lowest point several hours postoperatively. Concurrently both blood lactate and inorganic phosphate were increased.

6. That man can withstand up to 2 hours of severe respiratory acidosis (arterial pCO_2, 145 mm. Hg, and pH 6.9) is illustrated by 1 patient who made an uneventful recovery after restoration of normal acid-base balance.

7. There were 5 patients who died and 3 who were transiently hypotensive but ultimately recovered. The course of this group, when compared to those who made uncomplicated recoveries, suggests that in each instance respiratory inadequacy due to atelectasis or pneumonia took place. If the resulting hypercapnea (pCO_2 over 65 mm. Hg) and acidosis (pH below 7.25) and the moderate accompanying hypoxia were prolonged beyond a few hours, hypotension occurred. Circulatory failure and sudden death followed when this situation persisted for 24 hours or more.

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Discussion

DR. HERBERT C. MAIER, New York, N. Y.—Dr. Clowes and his associates have indicated the importance of studies which correlate the oxygen, carbon dioxide, and electrolyte alterations with the respiratory and cardiocirculatory changes in the individual patient. By such a means, a better understanding of the early etiologic factors in postoperative complications may be reached.

Too often undue attention is given to one facet without integrating the various chemical alterations that are present, together with the clinical features of that particular patient. I think for this reason that Dr. Clowes' studies are of particular value because, as you can see, he has tried to correlate them with the clinical side of the same patient.

He referred to the finding that decrease in the arterial oxygen saturation is more likely to occur with a lobectomy than a pneumonectomy. This seems to be due to the fact that blood continues to circulate through the remaining lobe on the side where a lobectomy has been performed, in spite of the fact that such a lobe ventilates poorly in the early postoperative period. In other words, you have a discrepancy between the circulation and the ventilation leading to moderate degrees of hypoxia. That situation does not exist in case of a pneumonectomy.

I should like to emphasize the finding that hypertension during operation was not a frequent finding, in spite of the presence of severe degrees of hypercapnia in some patients. As you know, in past years, anesthetists have often considered the development of elevated blood pressure as a sign of carbon dioxide retention. As is so well known now, there are no reliable clinical signs of carbon dioxide retention during operation. However, in the postoperative period, as has also been shown, hypotension is a common occurrence.

Finally, the case that he illustrated, with sudden death occurring on the tenth day, again emphasizes the fact that patients with prolonged moderate hypoxia and carbon dioxide retention may eventually have sudden death occur; so sudden that one thinks of pulmonary embolism and coronary thrombosis, when actually it is just the end result of prolonged chemical disturbance.

DR. GEORGE W. WRIGHT, Cleveland, Ohio.—The papers by Dr. Spencer this morning and the one just delivered contain many things that are of interest to the physiologically minded person. I hope the authors will forgive me if I do not discuss the particular points raised in their papers, but rather comment on what I think are some very timely implications of these two papers for those of us dealing with thoracic surgery.

The maintenance of body pH within a very narrow range is perhaps the most important requirement for the continuation of life. You need not be reminded, I am sure, of the tremendous importance of the respiratory apparatus in providing this constant pH concentration under the daily stresses of life. To be sure, the blood has some buffering power and the kidneys over a long period of time are very important for the regulation of pH, but for the hour-to-hour maintenance of this very essential requirement, the lungs are pre-eminent.

A patient who comes to us to be treated is commonly impaired in the respiratory apparatus, and then by our very attempts to help him we further impair the respiratory apparatus. In addition, and this is becoming more apparent as we deal with the severely crippled cardiac patient, the proper metabolism in muscle is not going on in individuals who have inadequate blood circulation, and as a result, the stresses in the direction of a metabolic acidosis are commonly greatly enhanced in some of the patients that we deal with. We are therefore in the very sticky situation of an apparatus that will not produce the required alveolar hyperventilation to take care of the increased stress in the direction of metabolic acidosis.

I think this has been very nicely demonstrated in both Dr. Clowes' and Dr. Spencer's papers. Dr. Spencer's paper points out the thing that I think is most important. If we wait until there are clinical evidences of this breakdown, we have waited much too long. What we would all like to know is the earliest evidence that the body is getting behind in its ability to compensate and maintain a normal pH. The key to the situation is obviously the carbon dioxide tension of the blood.

In so far as I know, there is no clinical sign that will let us know when carbon dioxide tension is beginning to become altered. If we had such a sign, I think that I would not be here making this comment at the moment, because you would then all be aware in plenty of time to try to do something about your patient and wouldn't be waiting until the end of the problem.

I think it behooves us, therefore, to introduce into our routine care of patients something that will give us the evidence that carbon dioxide excretion is not keeping pace with its production or with the production of other acid metabolites. There are methods available. I shan't take the time to go into that. It is true that they are somewhat time consuming and require some personnel. I would suggest, however, that we introduce into our routine postoperative care, and I say "routine" because few of us will do it on any other basis, some measure of the CO_2 tension in the blood in order that we can give these people respiratory assistance long before they are so exhausted as to require some kind of an external pump.

DR. JOHN H. GIBBON, JR., Philadelphia, Pa.—I wish not only to congratulate George Clowes on a very nice physiologic study but to ask him whether in any of these clinical cases there had been instances in which hypoxia and hypercapnia had not coincided, as they did in the patient that died on the tenth day.

About 2 years ago, in *The Journal of Clinical Investigation,* Holaday concluded from studies of patients with mild hypercapnia that respiratory acidosis produced metabolic acidosis. This had not been in accord with all our observations.' Last December, we reported before the Southern Surgical Association experimental studies in which we produced pure hypercapnia without hypoxia and pure hypoxia without hypercapnia. We found no evidence that metabolic acidosis, as evidenced by a decline in buffer base and an increase in lactic acid, occurred with hypercapnia, but these changes invariably occurred with hypoxia.

I well realize that metabolic and respiratory acidosis are both present in most patients postoperatively, as in the case Dr. Clowes presented. However, one condition does not produce the other experimentally. In fact, hypoventilation produces a fall in the blood lactic acid rather than a rise.

DR. CLOWES (Closing).—I think Dr. Maier's and Dr. Wright's points about the need for knowledge of the presence of hypercapnia and an understanding of the mechanisms which produce it are very pertinent. By way of therapeutic implications, it would appear, and I am afraid I didn't have time to go into this, that one should administer oxygen in questions of doubt to convert the patient of a mixed situation, if possible, into the purely acidotic one until something can be done about it. It appears that one can stand hypercapnia better in the presence of arterially saturated blood than otherwise.

I feel that there are two methods available to every surgeon who has access to any kind of laboratory. One is to stick needles into the arteries and simply measure the pH and then find out what the CO_2 combining power is and make an educated guess. If he can arrange to use the bubble technique of Reilly to measure the pCO_2 and oxygen tension, he is in good shape.

Dr. Wright drew to my attention, as we came out here the other day, that rebreathing—not exchanging but rebreathing—into an infrared analyzer will give a pretty good idea of what the CO_2 level in the arterial blood is. The lack of clinical signs of hypercapnia makes it very essential to know about this sort of thing.

Dr. Gibbon, I would agree with you completely on the matter of the difference in lactic acid production in the presence of hypercapnia. About 1955, I think, we presented a paper, also on dogs, distinguishing between hypoxia and hypercapnia, at the American Surgical Association, and our experience in that circumstance was exactly the same as yours. However, in the absence of hypoxia, lactic acid does rise considerably during operations, as many people have pointed out. This, in all probability, is more related to the depression of cardiac output

and to the inadequate perfusion given various vascular beds. On the other hand, I think that some emphasis should be made of the role of the liver in relation to lactic acid. A poor liver or a depressed one cannot deal with lactic acid as fast as the normal liver; therefore, perhaps the anesthetic, hypercapnia or hypoxia, has something to do with this situation.

I would say that we have not seen in the postoperative situation a clinical case that was purely hypercapnic. The average patient is slightly hypoxic, but his pCO_2 is perfectly normal.

PERICARDIECTOMY FOR RELAPSING PERICARDITIS AND CHRONIC CONSTRICTIVE PERICARDITIS

W. S. Blakemore, M.D., H. F. Zinsser, M.D. (by invitation), C. K. Kirby, M.D., W. B. Whitaker, M.D. (by invitation), and J. Johnson, M.D., Philadelphia, Pa.

DECORTICATION of the heart is widely accepted as the surgical treatment of chronic constrictive pericarditis. There remains some difference of opinion as to the surgical approach and the extent to which the pericardium must be removed.[1] Early reports of Délorme,[2] who described the pericardiectomy, and the more recent reports of Mannix and Dennis,[3] and Shumacker and Harris,[4] who advocate pericardectomy in patients with chronic effusion and cardiac tamponade, indicate the increasing acceptance of earlier operation. We present for consideration a proposal to remove the diseased pericardium before the scar contracts to the extent of reducing the efficiency of the cardiac mechanism. This is not proposed as a treatment for patients with acute pericarditis, but rather for those in whom the relapsing character of the disease has been established even in the absence of cardiac constriction or tamponade. Holman and Willett[5] recommended early surgery in patients with proved tuberculous pericarditis. The 4 patients which they reported had developed severe cardiac impairment prior to surgery, but they clearly recognized the desirability of early surgery and pointed out the ease of dissection when the effusion was present, in contrast to the difficulties encountered when the operation was delayed until the pericardium and myocardium were intimately fused. Antimicrobial therapy now provides considerable protection against dissemination of the disease following operations performed while the tuberculous process is still acute, and the fear of dissemination should no longer unduly delay operation in these patients.

For a number of years, we have performed pericardiectomy and decortication of the heart in patients with pericardial effusion in the absence of proved tuberculosis and prior to the time when they have classical findings of myocardial constriction. Among the many patients who have acute pericarditis, a few have the clinical findings which we believe can be recognized by rather typical symptoms in which the relapsing character of the disease is a prominent feature.

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Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

All of these patients have had an acute illness, usually easily diagnosed as pericarditis and associated with pleuritis and slight pleural effusion. Following symptomatic nonspecific treatment, the patients appeared to make a good recovery when the process was rekindled by physical exertion which was often mild. Chest pain, malaise, fever, pericardial friction rub, changes in electrocardiogram, leukocytosis, anemia, increase in the sedimentation rate, and reversal of the serum albumin to globulin ratio were commonly found. Progressive changes in the cardiac silhouette were noted by roentgenographic examinations (Table I). Repeated hospitalizations were required (Table II) and the patient approached

TABLE I. CLINICAL FINDINGS

Fever	Electrocardiogram changes
Pain	Leukocytosis
Malaise	Anemia
Weight loss	Increased sedimentation rate
Pericardial rub	Change in cardiac size by x-ray
Pleuritis or pneumonitis	

The patients with relapsing pericarditis had an initial acute episode easily diagnosed as pericarditis. These symptoms and laboratory findings were commonly found. The changes in the cardiac silhouette were not found in 1 patient and evidence of mild constriction was found in only 2 patients. In both of these patients, operative treatment had been recommended prior to the evidence of constriction.

cardiac invalidism accompanied by misery and despair.[6-8] The process was quenched by bed rest and medical therapy and, prior to recognizing the typical course of the disease, there was reluctance to recommend surgery for these patients. When they returned to their normal activity, the disease was reactivated and the process was repeated with greater irreversible damage. The loss of work

TABLE II. PREOPERATIVE HISTORY (8 PATIENTS)

		AVERAGE	TOTAL
Duration of symptoms	1.25-11.5 mo.	5 mo.	40 mo.
Hospital admissions	1-4	3	23
Per cent of time in hospital	20-100	50	(20 mo.)
Per cent of time at work	0-40	20	(8 mo.)

A prominent feature in the evolution of our recommendation of surgery for relapsing pericarditis was the prolonged invalidism with frequent hospitalization and inability to continue gainful employment from the time of the initial symptoms.

and livelihood was impressive, and only 20 per cent of the time from the onset of symptoms to the time of operation was utilized in gainful employment by these patients (Table II). Actually, only 2 patients were able to work during any part of the time following the onset of their symptoms and 1 of these continued as a salesman only by his extraordinary persistence, despite his constitutional symptoms.

Constriction or tamponade in a mild form was noted in only 1 of these 8 patients during a late phase and prior to the time we were recommending early surgical removal of the pericardium which was not overtly interfering with the cardiac function. In the earliest period, the operation was infrequently suggested and the suggestion poorly received by physicians. Most patients were known to recover from the acute episode of pericarditis regardless of the etiology.

Only in the unsual instance when the process of healing contracted the pericardium sufficiently to constrict the heart was surgical treatment accepted prior to the time when the relapsing character of pericarditis was recognized.

In another group of patients, the disease was insidious and the first attack of pericarditis was actually so benign that when constriction had developed, the diagnosis could not be made with certainty.[9] When calcification is present in the pericardium, the diagnosis of constrictive pericarditis is usually considered, but, when calcification is not present and the classical findings of constrictive pericarditis are not found, there is often a reasonable doubt of its presence in a patient with severe myocardial insufficiency. In a number of patients it has been necessary to do an exploratory thoracotomy through a left anterior intercostal incision to determine the presence or absence of pericardial disease.[11] Fortunately, in those patients on whom this procedure has been done, no deaths have occurred except in 1 of our first patients more than 10 years ago. In this patient, the incision was too small and the constrictive pericarditis which he had was unrecognized at the time of operation.

Among those patients in whom the diagnosis of chronic constrictive pericarditis is established, a prominent feature is often ascites. They frequently give a history that for many months or years their treatment has been directed with the diagnosis of ascites or cirrhosis of the liver while they continued as semi-invalids under the care of their internists. In patients with a moderately enlarged heart without pericardial calcification, it may be extremely difficult for the best cardiologists to be certain as to whether or not constrictive pericarditis contributes to the clinical findings. When the patient reaches the surgeon, the healing process has usually advanced and the pericardium is tightly attached to the myocardium. The surgical removal of the pericardium from the myocardium may be tedious and difficult. The patient is often in chronic cardiac failure, may have long-standing liver damage and ascites, and may be a poor surgical risk. Such a patient requires intensive preoperative treatment, a skilled and experienced anesthetist, and meticulous care to assist him through the period of surgical care. Among the 28 patients with chronic constrictive pericarditis whom we have treated, there have been 3 operative deaths. All of these deaths occurred in patients with long-standing heart failure. It would appear better to remove the diseased pericardium early in the course of the disease before the process is advanced, at a time when the patient's heart is not constricted and the patient is a better operative risk. At this time the removal of the pericardium, as well as the complete surgical care, is easier (Table III).

TABLE III. PERICARDITIS—OPERATIVE MORTALITY

	PATIENTS	DEATHS
Pericarditis, chronic constrictive	28	3
Pericarditis, recurrent nonconstrictive	8	0

Among the 25 patients with cardiac decortication for chronic constrictive pericarditis, there were three operative deaths compared with no operative deaths following operation for relapsing pericarditis. In the latter patients the operation is easier and morbidity is appreciably less. This operation is not recommended for acute pericarditis but it is indicated for relief of symptoms and disability incurred with the relapsing form of the disease as well as prophylactic in preventing the development of chronic constrictive pericarditis.

In the preoperative preparation of these patients, diuretics, digitalis, antituberculous therapy, and bed rest are utilized. Although the diagnosis of active tuberculosis is not proved, these patients are given antituberculosis therapy during the preoperative and postoperative periods until reports from pathologic and bacteriologic examinations are available to exclude the diagnosis of tuberculous pericarditis. Steroid therapy is probably not helpful.

The choice of approach in these patients has been individualized and, while wide exposure is desirable, the condition of the patient often does not permit the extension of the procedure that is necessary to obtain such exposure. In patients with constrictive disease, a transsternal incision is used.[10, 11] In very poor-risk patients or when the diagnosis is uncertain, the incision is started as a left anterior thoracotomy incision and, if indicated, is extended across the sternum into the right anterior thorax. In the patients with relapsing pericarditis, the midline sternal splitting incision provides adequate exposure.[11] The pericardium is resected widely and, whenever it is necessary, the epicardial layer is removed from the ventricles, although it is usually insignificant in extent. The operative area is drained via the pleural space. The pericardium has not been tightly fused with the myocardium and the operative procedure has proved to be easy. In the chronic cases of constrictive pericarditis in which the pericardium is more tightly adherent we use the transverse incision with wide exposure to both pleural cavities in the event that the cardiac cavity might be entered during the dissection. Rupture into the ventricle was the cause of death of 1 of our early patients in whom the exposure was inadequate to permit recovery from this catastrophe. The dissection should not be extended beyond the area where the exposure permits recovery. The thin-walled atria are especially vulnerable. The pericardium should be removed from phrenic nerve to phrenic nerve, the diaphragmatic surface, and, in most instances, it can also be resected on the left side posterior to the phrenic nerve. We have usually freed the pulmonary veins and venae cavae where they enter the pericardium but in no instance has sufficient constriction been found to be certain that obstruction at this point was a major factor in the disease. The removal of the constricting tissue over the ventricles appears to be the factor of greatest importance.

TABLE IV. POSTOPERATIVE HISTORY (8 PATIENTS)

		AVERAGE	TOTAL
Deaths	None		
Hospitalization	12-24 days	16.5 days	131 days
Returned to work	2-2.5 mo.	2.1	17 mo.
Per cent working	100	100	100

Among these patients who had pericardectomy and cardiac decortication for relapsing pericarditis, there have been no deaths, hospitalization after operation has been short, all the patients have returned to their previous work before 2½ months, and none has had a recurrence of his symptoms.

Except in those patients with evidence of constrictive disease, we have recommended the operation only to those patients with repeated acute episodes of the disease or to those who have reached the smoldering phase described earlier.[7] In no instance to date have any of the patients had recurrence of their pericarditis and, in each, the recovery has been rapid and apparently complete

(Table IV). The presence of pericardial fluid may also be an indication for early operation. We have recently had the opportunity to observe 2 additional patients. Each developed an acute febrile illness with massive pericardial effusion. In each, the large cardiac silhouette rapidly decreased in size and, in a period of 3 and 4 weeks, respectively, frank constrictive pericarditis was present. Although the operative procedure was not technically difficult in either patient the removal of the pericardium may have been easier if the procedure had been carried out earlier.

No specific etiology of the pericarditis has been found in any of the 8 patients with relapsing disease, and in only 4 of the 28 patients with chronic constrictive disease was it proved to be due to tuberculosis. The exact mechanism by which the operation terminates the febrile relapsing illness is not clear. The extensive removal of diseased tissue may eradicate the focus of infection, but this explanation is not entirely satisfactory because of the associated pleuritis and mediastinitis and since the posterior portion of the pericardium is not excised. The removal of diseased tissue accompanied by adequate drainage of the pericardium are undoubtedly additional factors. Button biopsies and windows in the pericardium[12] are less apt to provide adequate drainage and may seal over to give a recrudescense of the disease[8] and we do not consider these procedures adequate. We have not removed large portions of diseased pleura when it is found in patients.

In the postoperative period, overhydration is avoided and the patients are ambulated slowly. With the relief of their symptoms they may feel sufficiently improved to exercise without appropriate regard for the fact that the myocardium is usually involved in the disease process. For this reason they have been given a schedule of limited activity for the first month.

The results after pericardectomy in patients who have had relapsing nonconstrictive disease have been gratifying. All of the patients have returned to their original employment within 2½ months after operation (Table IV).

SUMMARY

1. Patients with constrictive pericarditis have often been permitted to remain in cardiac failure with secondary liver damage and ascites far longer than seems reasonable.

2. Decortication of the heart in the presence of chronic constrictive pericarditis may be difficult because of the technical problems imposed by fusion of the periardium and epicardium and because of the associated dysfunction which accompanies chronic cardiac failure.

3. Pericardectomy and decortication of the heart should be carried out before the patient becomes a poor surgical risk due to the deterioration associated with cardiac constriction and during the time when surgical removal of the pericardium is an easier procedure.

4. The pericardium in patients with tuberculous or idiopathic pericarditis and pericardial effusion should be removed without waiting for the development of constrictive pericarditis.

5. There is a group of patients who have recurrent bouts of nonspecific acute pericarditis who may suffer considerable long-term disability as a result of their disease. These patients should have a pericardectomy and cardiac decortication. In 8 patients with relapsing disease of this type, all have had excellent clinical results following operation.

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DISCUSSION

DR. JOHN F. HIGGINSON, Santa Barbara, Calif.—The notion of trying to prevent the rather horrendous end-result which we all have seen in advanced, chronic, long-standing constrictive pericarditis is commendable and this should be done, certainly, if we can possibly do it.

However, I was also attracted by what was in the abstract of the essayists' paper regarding difficulties in the diagnosis of some of these cases, despite intensive investigation preoperatively. In order to show one of the surprises that may be encountered at the time of exploration of a patient in whom suggestive findings are present, and who is not left unexplored for many years, I would like to show one slide and describe a patient who had all of the typical findings of congestive failure with all of the angiocardiographic and catheterization studies typical of constrictive pericarditis.

This 48-year-old woman had had 1 year of dyspnea on exertion with some ankle edema. These symptoms increased and progressed, and she finally developed anasarca with distended neck veins but with a quiet heart which was moderately enlarged and with an unremarkable electrocardiogram except for incomplete right bundle branch block and low voltage. The diagnosis was made clinically of unexplained myocarditis with myocardiofibrosis. She continued to develop more and more edema and then developed ascites. When she was first seen by Dr. Leonard Rose, of Portland, Oregon, he performed right heart catheterization on Nov. 14, 1957, and obtained the findings of narrow pulse pressure, very

high diastolic pressure in the right ventricle but with normal systolic pressure in the right ventricle and pulmonary artery. A marked characteristic dip and plateau were present, and fluoroscopy showed diminished cardiac pulsations.

I explored this patient on Dec. 3, 1957, and here [slide] you can see the enlarged heart that was encountered. The pericardium was perfectly normal. However, a very remarkable finding could be easily noted as one watched the heart. The ventricles came to a sudden, sharp, slapping termination of diastolic filling about halfway of what one would expect, quite obviously suggesting stiffening of the myocardium. A biopsy of the right ventricular myocardium was done with a Silverman needle, and a biopsy of the right auricular appendage was taken in the usual manner. The appendage and the right atrium were greatly dilated and enlarged.

Pathologic examination established a diagnosis of primary amyloidosis. Cardiac mural disease, therefore, is another explanation that must be taken into consideration whenever the clinical picture suggestive of constrictive pericarditis is present, despite highly suggestive findings by angiography and catheterization studies.

DR. SAFUH ATTAR, Baltimore, Md.—I would like to ask Dr. Blakemore three questions.

The first one is, what is the earliest time interval elapsing between the onset of the disease and the pericardiectomy among the 8 patients operated upon?

Second, will you operate on a patient with a clinical picture of pericarditis with a venous pressure of 300 which, on medical treatment, came down to normal, but clinically the patient persisted in having evidence of recurrent acute pericarditis, as done in your cases?

The third question is, in case the patient definitely has recurrent acute pericarditis, as shown in your cases, and does suffer of myocarditis, would you interfere early or would you wait until it goes to the chronic stage and then interfere?

DR. J. GORDON SCANNELL, Boston, Mass.—I should like to say how much I have enjoyed this presentation and to emphasize a point which was mentioned; namely, the importance of doing a liberal decortication of the ventricle at the time of the initial procedure.

We have had the opportunity to see a few late recurrences among the very early cases of Dr. Churchill, and also recurrences in 1 or 2 patients operated upon during a relatively acute phase and a radical decortication not done. In these recurrent cases, the source of the difficulty has invariably been on the posterior aspect of the left ventricle, in the region of the junction between the left atrium, the left pulmonary veins and the atrioventricular groove. Had we always been insistent on getting away back to this point at the original operation, when it is relatively easy to do through a left anterolateral approach, especially in a subacute phase of the disease, I am quite certain that this recurrence could have been avoided.

Constrictive pericarditis, as you know, seems to be a disease primarily of the pericardium itself, not the pericardial cavity, and it is important to resect the pericardial sac, not merely just free up the heart.

DR. EDGAR P. MANNIX, Manhasset, Long Island, N. Y.—I think Dr. Blakemore is to be congratulated for his excellent presentation in drawing our attention to not only the very important clinical entity of chronic constrictive pericarditis, but also, what is more important, I believe (because it is less well understood), the place for the surgical treatment of chronic pericarditis during the stage of effusion.

Patients with the latter type of problem are most difficult to acquire. In my experience, internists are reluctant to advocate surgical treatment for patients suffering with chronic pericarditis with effusion, yet, I am sure, as Dr. Blakemore will agree, that the operation is much easier to do during the stage of effusion and the deleterious effects of chronic constriction can be avoided.

This point, of course, has been made many times in the past. Dr. Emile Holman more than 5 years ago, as a matter of fact, advocated pericardiectomy during the acute phase of tuberculous pericarditis. Dr. Barker, among others, from the University of Michigan, has reported a case of chronic pericarditis with effusion which lasted for as long as 6 years and then went on to constriction.

I would like to close my comments by simply pointing out that in keeping with Dr. Higginson's comments, the diagnosis is very important. We believe it is important to exclude pericarditis with effusion when it is on a rheumatic basis, because this almost always means active rheumatic carditis. In our opinion these people should not be operated upon at that time.

There is another entity that occurs in small children and infants which is very difficult to diagnose, sometimes accompanied by pericardial effusion, namely, the idiopathic chronic myocarditis. There are other medical entities; myxedema with pericardial effusion, for example, is one which can confuse the diagnostic picture and which can be treated medically by thyroid medication.

DR. EMILE HOLMAN, San Francisco, Calif.—This presentation has pleased me very much as it recognizes the need of a radical pericardectomy when operating for constrictive lesions.

I would like to place on record a patient who had been operated upon three times for constrictive pericarditis, and who still had his ascites. He remained in a Marine hospital for over a year, having his abdomen tapped periodically for recurring ascites.

We operated upon him again with great reluctance, and in the course of the operation we accidentally entered the right ventricle, and had considerable difficulty extricating ourselves from that situation. Because of the patient's poor condition, I was unable to remove any pericardium. I did, however, incise the pericardium directly over the inferior vena cava, spreading the edges of the thickened pericardium for about 2 cm., and did the same over the superior vena cava.

This patient has not required tapping since that operation, indicating the great importance of liberating all critical areas, in this case the inferior and superior venae cavae, which had escaped adequate liberation at the three previous operations.

DR. BLAKEMORE (Closing).—I believe the questions can be divided into two groups corresponding to the separation we have made in the presentation. This may avoid confusion, since we have discussed a form of pericarditis which we have called relapsing pericarditis and also discussed many of the difficulties which have occurred in the diagnosis and treatment of chronic constrictive pericarditis.

Dr. Higginson's comments again emphasize the difficulties of establishing the diagnosis in patients with chronic constrictive pericarditis. We have found that excellent cardiologists with the benefit of modern diagnostic techniques have been unable to eliminate the diagnosis of constrictive pericarditis in patients who are severely ill and have congestive heart failure of unknown etiology. In these instances, an exploratory operation is often necessary diagnostic procedure. The earliest time that we have been able to definitely recognize the signs and symptoms of relapsing pericarditis has been 6 weeks, and we were able to do this only in 1 of the latest patients whom we have seen. The principal point in making the diagnosis of relapsing pericarditis is to recognize the smouldering phase in a patient in whom the disease can be rekindled by physical activity, can be quenched by bed rest and medical therapy, and reactivated as physical activity is again increased. Dr. Harry Zinsser, a medical cardiologist in our group, has been speaking to groups of internists, and as this type of disease is better recognized the surgeons will see many of these patients earlier in the course of disease.

Dr. Attar asked a question about a specific patient who had increased venous pressure and recurrent episodes of pericarditis. I believe this is a patient in whom we would recommend removal of the pericardium. If we could recognize the relapsing form of the disease early in its course, we would recommend an early operative procedure. However,

the operation is not to be recommended for every patient with acute pericarditis. Chronic constrictive pericarditis may in contrast follow an unrecognized episode of acute pericarditis, and an earlier operation would be desirable for these patients prior to the onset of the severe myocardial changes which accompany long-standing constriction.

We are honored to have Dr. Holman discuss this paper since his earlier publications have strengthened our position in advocating early operative procedures for our patients. With regard to the patients which Dr. Holman mentioned, we are in agreement that no operative procedures are indicated for the treatment of pericarditis of rheumatic origin, but pericardiectomy is indicated for the patients with idiopathic relapsing or tuberculous pericarditis. In these patients, early operation is desirable before extensive myocardial damage occurs which significantly increases the operative risk. Dr. Holman and Dr. Scannell advocate extensive resection of the pericardium, and we are again in agreement. We make some reservation and present that, in certain patients who are severely ill and in whom the operation of this magnitude increases the operative risk, the procedure can be shortened. The diseased pericardium about the atria and veins is left, but the pericardial and epicardial layers are removed from the ventricles.

Délorme, who utilized the cardiolysis procedure and described the principles of the pericardiectomy operation, was critical because the operation did not remove the epicardial layer. Many patients require the removal of this layer as well as the pericardium, but the epicardial layer has not been of significant thickness in some patients whom we have treated with relapsing pericarditis to warrant removal.

THE RESULTS OF SURGICAL CORRECTION OF ATRIAL SEPTAL DEFECT COMPLICATED BY PULMONARY HYPERTENSION

Harold V. Liddle, M.D. (by invitation), B. W. Meyer, M.D., and John C. Jones, M.D., Los Angeles, Calif.

ATRIAL SEPTAL DEFECT of the secundum type is the most successfully managed intracardiac defect to which surgical attention has been turned. In the last 10 years, a variety of surgical techniques has been devised for its closure. In 1948, Murray[1] described obliteration of the defect with fascia lata strips. Later, Gross,[2] Bailey,[3] and Sondergaard[4] described blind operative procedures which have been very successfully applied.

Presently, open heart methods make possible direct, accurate suture of the anomaly with low mortality. The majority of patients with secundum atrial septal defects have normal or minimally elevated right ventricular and pulmonary artery pressures. This defect, unlike ventricular septal defect, is almost never complicated by pulmonary hypertension in children. Approximately 15 per cent of these patients in adolescence and adulthood do have pulmonary vascular obstruction. Repair of the defect in the presence of severe pulmonary vascular obstruction increases the morbidity and mortality tenfold.

PHYSIOLOGY OF ATRIAL SEPTAL DEFECT

The physiologic characteristics of secundum atrial septal defects are unique. At birth there is little flow across the atrial defect, but as systemic resistance increases a left-to-right shunt develops. The volume of this shunt gradually becomes larger and results in pulmonary flow which may be two to three times systemic flow. To accommodate this volume of blood, the pulmonary vessels dilate and their resistance falls. The right ventricle becomes overloaded by this increasing burden, particularly during the diastolic phase. It dilates in response to the demand, and the electrocardiogram reflects this dilatation as delayed conduction. Prolonged exposure to excessive flow is occasionally associated with morphologic changes in the pulmonary vessels resulting in pulmonary hypertension. In the presence of severe pulmonary vascular obstruction, the volume of shunt is reduced but remains predominantly left to right.[5, 6]

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Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

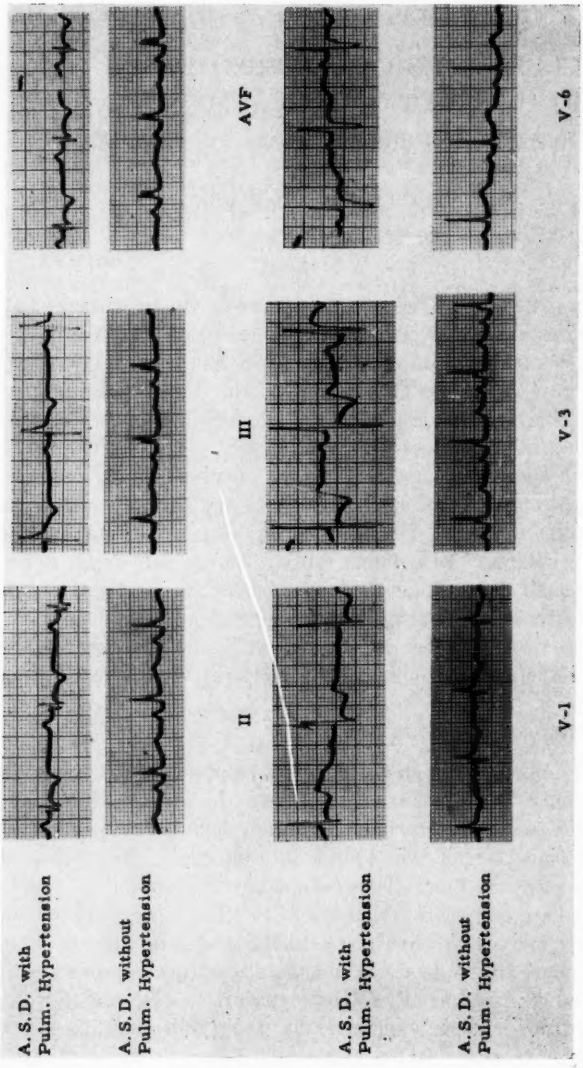

Fig. 1.—Electrocardiogram in atrial septal defect, with and without pulmonary hypertension.

Even with a reduction of shunt, right heart work actually increases as pulmonary resistance increases. Closure of an atrial septal defect, complicated by severe pulmonary vascular resistance, is often associated with acute right ventricular dilatation, fibrillation, and death. Selection of patients with severe pulmonary hypertension who will tolerate closure of the defect is difficult, and evaluation of pulmonary artery pressure alone is not a sufficient criterion.

REPORT OF CASES

During the last 5 years, over 180 patients with atrial septal defect of the secundum type have been surgically treated in our unit. One hundred forty of these patients, who had normal pulmonary artery pressures, were operated upon without mortality. We consider pulmonary artery pressures in excess of 50 mm. Hg to constitute pulmonary hypertension. The 15 patients in this series with simple secundum atrial defects, complicated by pulmonary hypertension, form the basis of this report. Their ages ranged from 16 to 56 years, an average of 39 years. There were 11 women and 4 men.

Symptoms.—These patients all exhibited progressive dyspnea and fatigue. Two had recurrent hemoptysis. Four patients developed chronic atrial fibrillation, and 2 had been in congestive failure. Half the patients suffered chest pain. Cyanosis at rest was present in 4 patients. One patient had previously undergone exploration with a mistaken clinical diagnosis of mitral stenosis. The 16-year-old girl had very advanced kyphoscoliosis and pulmonary fibrosis. The progression of symptoms in these patients was characteristically rapid, an important event in evaluating the risk in these patients. Cardiac physical findings revealed cardiomegaly and right ventricular heave in all but 2 patients. There was a loud systolic murmur heard along the left sternal border in the third to the fifth left intercostal spaces. Seven patients had a blowing diastolic murmur in the third to fourth left intercostal spaces. The second heart sound was, in each case, markedly accentuated and widely split.

X-ray Findings.—Preoperative x-ray studies of the chest of each patient were reviewed, and the cardiac silhouette was of normal size in 2. Cardiomegaly in the remaining patients varied from slight to severe. The main pulmonary artery shadow was enlarged in all cases—strikingly so in 4. Hilar vessels were enlarged in every case. The relative sizes of peripheral and hilar vessels were then compared. Instead of the peripheral engorgement usually associated with atrial septal defect, these patients exhibited disproportionate diminution in the caliber of peripheral vessels when compared to the hilar vessels. In the x-ray studies of patients who died at operation, heart size was not particularly increased, but the pulmonary artery segment was exceedingly large. The discrepancy between peripheral and hilar vessel size was most marked in these individuals. Cardiac size could only occasionally be correlated with the degree of pulmonary hypertension.

Electrocardiographic Findings.—Electrocardiograms revealed right ventricular hypertrophy in all but two instances (Fig. 1). One of these 2 patients

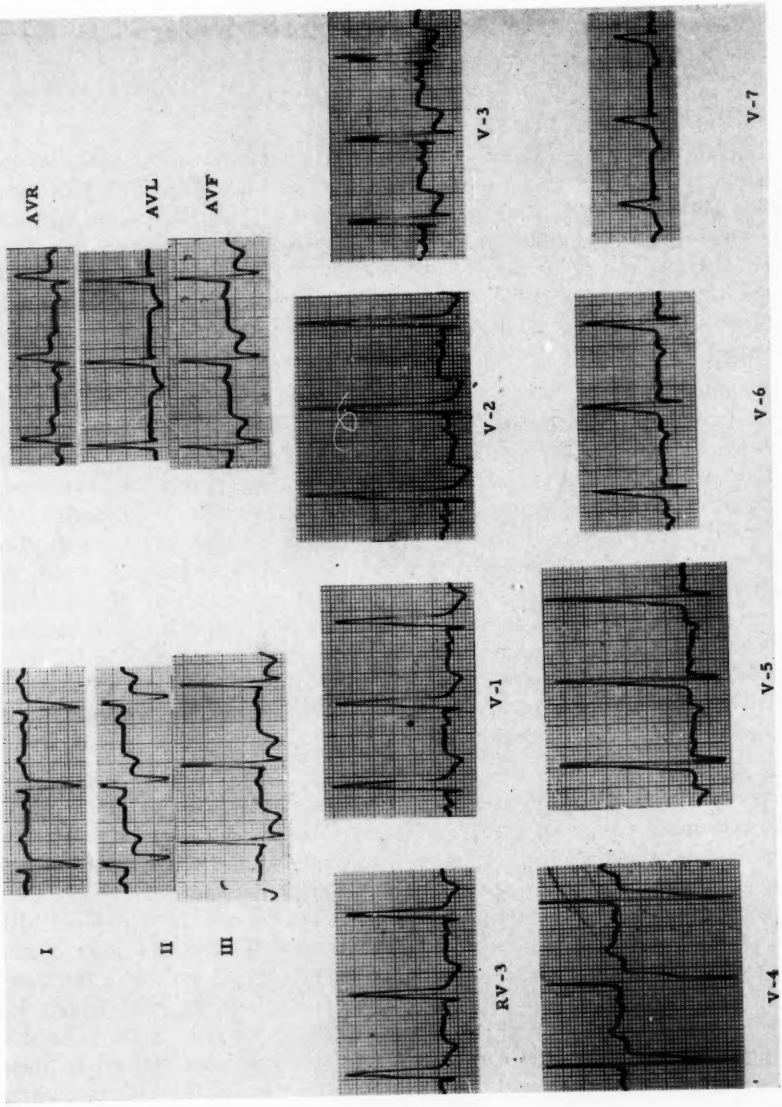

Fig. 2.—Electrocardiogram in atrial septal defect with severe pulmonary hypertension.

had a pulmonary artery pressure of 100, but right ventricular hypertrophy was *not* evident in either the electrocardiogram or the roentgenogram. In the second patient, the electrocardiogram showed only low voltage and atrial fibrillation. Electrocardiograms in the remaining patients showed moderate to severe right ventricular hypertrophy. In the precordial leads, the RSR′ pattern was consistently seen. The secondary R wave was significantly tall in some of these patients. In 6 of the 7 patients who died either because of irreversible pulmonary vascular change, or who were not benefited by surgery, the electrocardiogram showed strikingly tall secondary R waves in V_3R and V_1 (Fig. 2). Only 1 of the patients who fared well had a similar pattern, and she had chronic pulmonary disease.

Catheterization Data.—Cardiac catheterization was performed in all patients prior to operation. Right ventricular and pulmonary artery pressures were in excess of 50 mm. Hg in every case (Table I). Pulmonary artery pressures were between 50 and 70 mm. Hg in 7 patients. In 2, it was between 80 and 90 mm. Hg; in 6 it was between 93 and 105 mm. Hg. Pulmonary artery pressure never exceeded systemic arterial pressure, even in instances of the most severe pulmonary vascular obstruction.

TABLE I. RIGHT HEART CATHETERIZATION (PREOPERATIVE DATA)*

PATIENT	AGE	SEX	SUP. V.C.	R.A.	R.V.	P.A.	B.A.	P.V.	PULMONARY FLOW	SYSTEMIC FLOW
D. B.	33	M	*49*	2 *47*	82 *69-72*	84-107 *67*	110/49 *88*	*94*		
R. B.	37	F	7/2 *60*	14/6 *68-74*	80-110/0 *73-75*	100/50 *76*	140/90 *87*		2 × systemic	
M. L.	39	F	*63*	4/2 *82*	67/2 *85-87*	63/28 *85-87*	116/64 *95*	*100*		
D. S.	25	F	*52*	12/8 *59-62*	80/6 *62*	80/30 *62*	110/80 *96*			
T. R.	35	F	*66*	8/4 *78*	93/3 *78*	93/33 *75*	102/59 *93*			
P. S.	33	F	*51*	13/7 *66*	93/8 *70*	93/30 *72*	 *92*			
F. J.	42	F	*63*	12/3 *89*	105/5 *91*	105/30 *90*	98/58 *98*	20/2 *97*		
D. M.	31	M	*59*	5/4 *62*	60/3 *70*	60/30 *70*	 *79*			
K. B.	48	M	*52*	7/1 *77*	60-78/4 *77*	53-78/27 *76*	110/70 *81*	9/3 *93*		
P. L.	41	M	*56*	14/8 *66*	100/8 *66*	102/37 *66-70*	148/87 *79-84*			
M. S.	42	F	*49*	20/13 *75*	50/12 *77-82*	55/23 *79-80*	94/53 *83*	*94*		
D. H.	52	F	*58*	8/3 *76*	60/3 *78*	62/30 *72*	142/80 *89.5*	*94*	5.74	4.43
J. C.	16	F	*63*	2 *83*	62/3 *84*	57/21 *84*	96/76			
J. B.	23	F	*69*	7/2 *83*	62/3 *83*	59/29 *83*	 *89*	*99*		
M. H.	56	F	14/10 *76*	25/3 *78-82*	100/5 *78*	100/39 *78*	110/78 *87*			

*Pressures and oxygen saturation (the latter in italics).

B.

A.

Fig. 3.—*A*, A subpleural small artery, with medial hypertrophy, shown emptying into a glomeruloid structure. *B*, High-power detail of the same vessel showing the glomeruloid structure and vascular lake emanating from it.

Changes in oxygen saturation at the atrial level were often at the lower limit of acceptability to make the diagnosis, which indicates that the left-to-right shunt was reduced in these individuals.

Flow and resistance calculations were not regularly made, so these data are unavailable.

Operative Technique.—The techniques employed for closure of these defects have changed as newer methods were described. The Sondergaard circumclusion operation was done in 2 patients. Three had atrioseptopexy, and 10 were closed by direct vision employing hypothermia. In all hypothermic procedures, the temperature was maintained in the range of 27° to 29° C., and the average period of circulatory occlusion was 7 minutes.

RESULTS

The immediate survival of patients following closure of atrial septal defect in the presence of pulmonary hypertension is largely predicated upon the ability of the right ventricle to overcome existing peripheral resistance in the lung. Long-term survival demands reversibility, or at least arrest, of the changes occurring in the pulmonary bed. The results in this study are recorded on that basis.

Immediately upon closure of the defect in 4 patients, the right ventricle dilated acutely, and fibrillated. These hearts could not be restored, even after re-opening the defect. A fifth patient satisfactorily tolerated closure of the defect, but at the conclusion of the procedure, the left ventricle ruptured. Pathologic examination revealed the left ventricular muscle to be 4 mm. thick, with marked fatty infiltration. We agree with Kirklin[7] that left ventricular failure is an unlikely complication in these patients. However, the transient increase in left auricular pressure, as shown by Morrow[8] and Hickler and Goodale,[9] was apparently sufficient in this pathologic ventricle to rupture it.

There are 2 patients who, although they survived, were not helped by correction of the defect. One of these patients lived for 3 years with progressive pulmonary vascular disease, cardiac failure, and constant cyanosis. Recatheterization 3 years postoperatively revealed rise of right ventricular and pulmonary artery pressures from 60 to 80 mm. Hg. He died of intractable failure, and at postmortem the defect was found to be completely closed. We believe that peripheral unsaturation persisted via shunts in his lung.

The second patient has survived for 3 years, but he is a cardiac cripple in chronic cardiac failure, mildly cyanotic, and with recurrent hemoptysis. Recatheterization 1 year postoperatively revealed minimal increase of pulmonary hypertension, but no evidence of shunting within the heart.

Serial sections of the lungs of 3 nonsurvivors were made, and they revealed microscopic arteriovenous communications at the small artery level and occasionally the arteriolar level (Fig. 3). These communications entered glomerular-like structures, which in turn emptied into large vascular lakes. The lakes

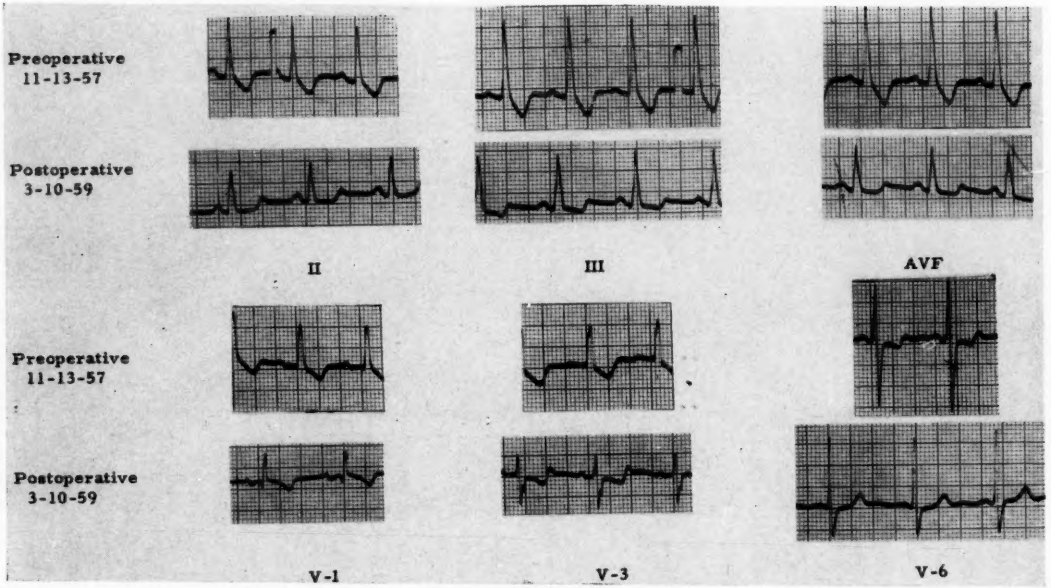

Fig. 4.—Pre- and postoperative electrocardiograms in reversible pulmonary hypertension.

were seen occasionally to follow alveolar septa, but more often to drain directly into veins. There is divided opinion in the literature[10-13] about the function of these structures. We are inclined to feel that they may be arteriovenous shunts, because they were seen in a patient who was proved at autopsy to have a healed septum, yet he had been deeply cyanotic for 3 years after surgery.

Eight patients comprise the second group. These individuals tolerated closure of their defects satisfactorily. One died 8 hours postoperatively of hemorrhage from an unrecognized bleeding internal mammary artery. A second patient who survived, has had a re-opening of the atrial septal defect and pulmonary hypertension has progressed. This defect had been closed by the Sondergaard method. The 6 remaining patients have progressed very well, with minimal or no limitation of their activity. Five have been followed closely at our institution, and in each there has been decrease of cardiac size, of hilar shadows, and of the second pulmonary heart sound. The sixth patient is reported by her family physician to have comparable improvement.

Four of these patients have been recatheterized from 1 to 3 years postoperatively (Table II). Each has had significant decrease in pulmonary artery pressure. In 1 of these, the right ventricular and pulmonary artery pressures fell from 100 to 50 mm. Hg (Table III), and the electrocardiogram showed reduction of right ventricular hypertrophy (Fig. 4). The pulmonary artery pressure in a second patient fell from 57/21 to 18/4, and the electrocardiogram showed only minimal evidence of right ventricular hypertrophy. This patient still has respiratory limitation due to kyphoscoliosis.

TABLE II. POSTOPERATIVE CATHETERIZATION DATA*

PATIENT	AGE	SEX	SUP. V.C.	R.A.	R.V.	P.A.	B.A.	FLOW	RESULT
D. M.	31	M		15/9	81/9	77/35	93/62		Dead 3 yr. PO in heart failure
			70	*64*	*63*	*59*	*86*		
P. L.	41	M		5/-1	73/0	73/27	128/67		Good—4 yr. PO
			69	*70*	*72*	*72*	*89*		
D. B.	33	M	9/7	9/7	93/8	93/88	110/70		Progressive deterioration —3 yr. PO
			77	*75*	*75*	*75*	*75*		
R. B.	37	F	10/5	10/5	55/10	55/30	138/80		Excellent—2 yr. PO
			76	*77*	*77*	*77*	*95*		
M. L.	39	F		6/0	45/0	44/18	128/60	*3.7* P.A.	Fair—2 yr. PO
			66	*69*	*67*	*66*	*90*	*3.4* systemic	
J. C.	16	F		2/-2	18/0	18/4	108/60		Excellent—4 yr. PO; kyhoscoliosis severe
			65	*68*	*67*	*68*	*94*		
J. B.	23	F	9/3	9/4	70/7	75/25	103/67		Recurrence of defect
			64	*69-83*	*73-85*	*75*	*88*		

*Pressures and oxygen saturation (the latter in italics).

In another patient, pulmonary artery pressure, 16 months after operation, had only fallen from 100 to 70 mm. Hg. Now, 4½ years after operation, pulmonary artery pressure remains high. There is no evidence that the defect has re-opened, but the patient remains cyanotic with exercise, possibly due to intrapulmonary shunting. Subjectively, he remains well after return to normal activity.

TABLE III. CATHETERIZATION DATA (R. B., 37, F)*

	SUP. V.C.	R.A.	R.V.	P.A.	B.A.	WEDGE	FLOW
Preoperative data	7/2 *60*	14/6 *71*	100/0 *74*	100/50 *76*	140/90 *87*	10/3	P.A. 2 × systemic by RISA
Postoperative data	10/5 *76*	10/5 *77*	55/10-0 *77*	55/30 *77*	138/80 *95*		

*Pressures and oxygen saturation (the latter in italics).

The electrocardiogram in a fifth patient, who would not be recatheterized, shows disappearance altogether of evidence of right ventricular hypertrophy, and symptomatically she is completely normal.

In summary, of the 15 patients with severe pulmonary hypertension who underwent closure of the defect, 5 died at operation. Two patients were unimproved, and 1 died of an operative complication. Six patients have had long-term survival and show regression of pulmonary vascular resistance. There has been recurrence of the defect in 1 patient.

DISCUSSION

Atrial septal defects are uncommonly complicated by advanced changes in the lesser circulation. Even among adults, pulmonary vascular obstruction is only occasionally present. This indicates that flow alone is not responsible for its appearance.

The size of the defect does appear to be related to the appearance of pulmonary hypertension. In this series, the defects varied in size from 3 to 7 cm., the average being 5 cm. We have had no patients with small defects who have had pulmonary hypertension.

The extreme increase in mortality associated with pulmonary vascular obstruction makes selection of patients for surgery with this complication critical. In this series, mortality was increased from zero in uncomplicated cases to nearly 50 per cent in those with severe pulmonary vascular obstruction. However, the fact that some of these poor risk patients can be permanently rehabilitated compels us to find some means of selection. Cyanosis is an indication of excessive right atrial pressure, and suggests irreversible pulmonary changes. Three of the 4 cyanotic patients in this series either died or were not improved by surgical intervention. A history of congestive heart failure predicted an unfavorable outcome. The degree of pulmonary hypertension in this study was, however, unrelated to the surgical result. Two of 5 patients who died at operation had pulmonary artery pressures below 65 mm. Hg, yet 3 patients who have done well had pressures between 93 and 105 mm. Hg. Neither the duration of symptoms nor cardiac size were related to survival.

Although no single or combination of data are incontrovertible evidence for selection of probable survivors, review of this series does suggest four factors which may be helpful guides. The first two, as mentioned above, are cyanosis and a history of cardiac failure. Patients with this combination will probably not survive operation.

An additional factor is a marked discrepancy between the caliber of hilar and peripheral vessels in x-ray studies. In this series, the combination of huge hilar vessels and peripheral starving was usually associated with irreversible vascular change. Although the peripheral vascular shadows were decreased in all these patients, there was not an unusual discrepancy in vessel size among the individuals with good results.

The electrocardiograms of 13 of these 15 patients showed right ventricular hypertrophy. Six of the 7 patients with irreversible pulmonary vascular change had severe right ventricular hypertrophy. In each case, the secondary R wave in V_3R and V_1, and the S wave in V_6 were strikingly increased. With the exception of the 1 patient who had advanced kyphoscoliosis, this magnitude of electrocardiographic change was not seen in the survivors.

SUMMARY

Simple atrial septal defects of the secundum type have been surgically treated in more than 180 patients in our unit. Of this number, there are 15 patients, all adult, whose defects were complicated by pulmonary artery pressures in excess of 50 mm. Hg. Five of these patients succumbed at operation, and 2 were not benefited by closure of the defect. A review of the charts of these 15 patients suggests four clinical and laboratory observations which are related to the surgical failure in 7 patients. They are the presence of cyanosis, the history of congestive heart failure, severe right ventricular hypertrophy, and a marked discrepancy in vessel size in chest x-ray studies. When two or more of these observations are made in a patient, there probably exist irreversible pulmonary vascular changes.

There is evidence that acetylcholine injected in the pulmonary artery may delineate its capacity for dilation. Theoretically, acetylcholine and cineangiography in combination may vastly improve our evaluation of the pulmonary vascular bed.

The very serious risk and relatively poor results of surgery in this group of patients, compared to the uncomplicated atrial defect, are urgent evidence that these defects should be closed when they are recognized. There is nothing to be gained by procrastinating, and none of these people will improve with time.

We wish to express our appreciation to Donald C. Fyler, M.D., Assistant Professor of Pediatrics, University of Southern California Medical School and Director of Cardiology Diagnostic Laboratory of the Childrens Hospital Society of Los Angeles, and to H. G. Peterson, M.D., Assistant Clinical Professor of Radiology, University of Southern California Medical School and Chief Radiologist of the Childrens Hospital Society of Los Angeles, for their help in assimilation of the data presented.

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DISCUSSION

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—The authors should be congratulated on this paper. I am wondering whether or not in the treatment of pulmonary hypertension with atrial septal defect they have considered or would care to discuss the problem of using a patch with perforations in it, and whether or not they would feel that this might obviate the sudden strain on the right ventricle.

DR. MEYER (Closing).—We have not used a perforated patch to reduce the size of the defect for we have felt that partial closure does not reduce pulmonary hypertension. This belief is based on catheterization data following partial closure of high pressure ductus and interatrial defects with very high pressures. We believe that if there is any release or reversibility of the pulmonary vascular obstruction, it can be determined at the operating table by the way the right heart reacts after defect closure. Invariably, with a fixed resistance in the lungs, the right heart will dilate and fibrillate after the defect is closed. In these patients, the defect had to be re-opened before normal cardiac function could be restored.

COARCTATION OF THE AORTA IN INFANTS

A Clinical and Experimental Study

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THE CUMULATIVE experience in the surgical management of coarctation of the aorta now spans a period of 15 years. It is well established that surgical removal of the constriction with restoration of continuity is the treatment of choice. In older children and young adults, operation can be done at very low risk, with every assurance of cure. Coarctation in infancy, on the other hand, is a less well-understood entity, and a number of formidable problems and confusing issues still exist. With the development of the flush technique for simple and accurate determination of blood pressure in newborn infants, it has become apparent that the lesion is by no means as rare as once thought, and, indeed, when found in combination with patency of the ductus arteriosus, is one of the leading causes of cardiac failure in early infancy.[1] As more cases are documented, it has been realized that anatomic classifications of coarctation which stress infantile versus adult forms are no longer tenable, for the adult form can cause trouble at 2 weeks of age, and the infantile form conversely can go undiagnosed until adulthood. A division on the basis of segment length into long and short segment types is also not too helpful, for one type merges imperceptibly into the other. A more useful classification simply states the relationship of the coarctation to the ductus (preductal, juxtaductal, or postductal) and whether the ductus is open or closed.[2] The association of coarctation in infancy with other cardiovascular malformations, particularly patent ductus arteriosus and ventricular septal defects, is well known, but the importance of patent ductus as a cause of difficulty has not been sufficiently stressed. There are differences of opinion among pediatric cardiologists as to the best method of management of these cases. Nadas[3] feels that a number can be brought out of cardiac failure and carried along until they are several years of age. Calodney and Carson,[4] and Goldring and his associates[5] have stressed the uniformly poor results they have had with medical management, and this led them to urge early surgical intervention. In spite of the prevalence of this lesion in the infant age range, there are relatively few reports of successful surgical treatment. Isolated cases have appeared[6, 7] and the papers of Mustard[8] and Smeloff[9] and their colleagues have included a small group of operated cases. A greater degree of success

Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

should be possible as more information is gathered concerning the basic as well as the clinical aspects of the disease. This report is an analysis of the total surgical experience at the St. Louis Children's Hospital with coarctation in infants (24 months of age and younger), together with experimental investigations concerning the physiologic derangements associated with coarctation and patent ductus arteriosus.

CLINICAL MATERIAL

Clinical experience with these seriously ill infants since 1940 now totals about 60 proved cases, and only 2 patients have survived beyond infancy on medical therapy alone. This prompted the policy of advocating early surgical intervention. The first operative repair was undertaken in March, 1949 (T. H. B.). Since that time, 16 infants under the age of 24 months have

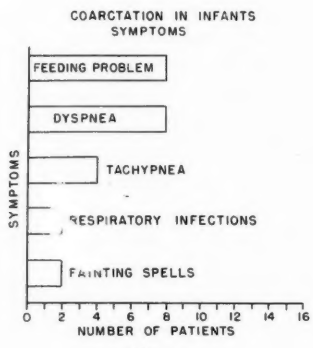

Fig. 1.

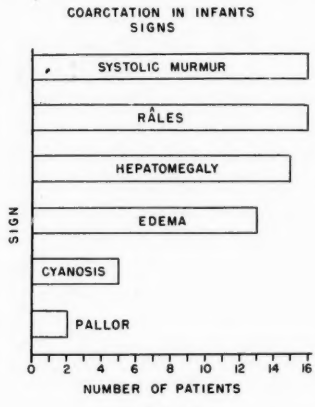

Fig. 2.

Fig. 1.—Symptoms encountered in the 16 infants in this series.
Fig. 2.—Physical signs present in the 16 cases.

been operated upon. (By comparison, during the same 10-year period, 72 patients over the age of 2 years have been repaired.) There were 11 males and 5 females. The ages on admittance ranged from 9 days to 24 months, but the majority of patients developed difficulty soon after birth. Thirteen of the 16 cases were under 4 months of age. The median age at the time of operation was 6 weeks. Symptoms and signs which brought the infant to the hospital were almost all related to cardiac failure, and as such were not helpful in making a specific diagnosis (Fig. 1). Most frequent were problems of feeding, dyspnea, and tachypnea. Physical examination revealed the signs of overt cardiac failure in all cases, with pulmonary congestion, hepatomegaly (1 to 5 fingerbreadths), and edema (Fig. 2). Five infants had cyanosis which cleared under treatment, and therefore was thought related to circulatory failure. Differential "girdle" cyanosis, supposedly typical of this condition, was not seen. All of the babies had a systolic murmur, graded 4 plus in 4, barely audible in 4, with intermediate gradations in the remainder. None

had a continuous murmur. Roentgenographic examination of the chest uniformly showed the same picture—a markedly enlarged heart with pulmonary vascular congestion and engorgement compatible with a left-to-right shunt and congestive cardiac failure (Fig. 3). The electrocardiograms were grossly abnormal. Right ventricular enlargement was present in 9 cases, left ventricular enlargement in 7 cases. A mixed group of 9 children also had myocardial disease. None of the patients had cardiac catheterization, and in only 1 infant was an intravenous angiogram done. This policy was dictated by the critical state of these infants, since it was felt that they would not withstand anesthesia for a diagnostic procedure.

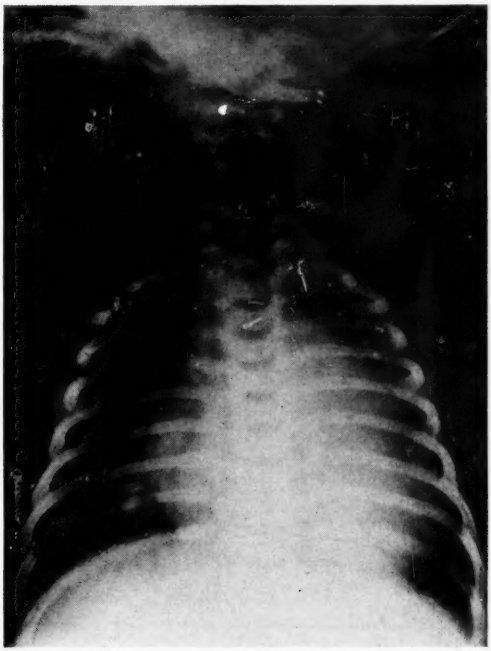

Fig. 3.—Roentgenogram of an infant with coarctation and patent ductus proved at autopsy. There were no other anomalies. The enlargement and configuration are typical of all the cases in this series.

The single most important examination was the determination of the blood pressure. The flush technique, utilizing the principle of reactive hyperemia, was first conceived and used in 1950, and the method standardized by 1952[10] (Fig. 4). Since that time it has become a routine part of the workup, and several of the infants included here were quickly diagnosed, having been admitted as feeding problems, etc. When the pressures are thought to be abnormal, a number of determinations are made by each of several observers over several days, and the results averaged. To be significant, the gradient must exceed 20 mm. Hg. In recent cases, flush pressures have been corroborated by an electronic pulse recorder (Infraton). In all the infants except

the first 2, the diagnosis was made by the flush technique. Fig. 5 shows the spread and the average values obtained. A mean gradient of 86 mm. Hg is indicative of the severe degree of coarctation present in these infants.

The average time lapse from admission to operation was 2½ weeks. All the babies showed a temporary favorable response to digitalis. Prior experience had taught that this improvement should not raise false hopes about

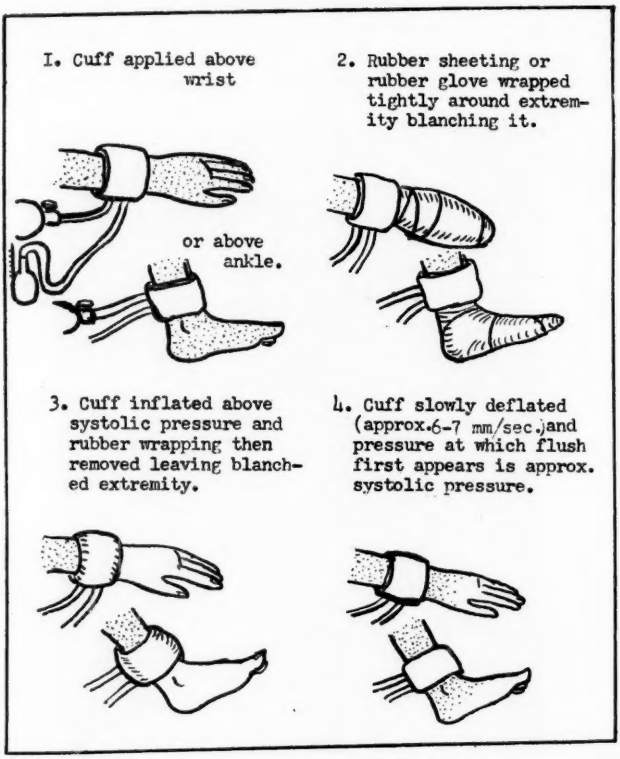
1. Cuff applied above wrist
or above ankle.
2. Rubber sheeting or rubber glove wrapped tightly around extremity blanching it.
3. Cuff inflated above systolic pressure and rubber wrapping then removed leaving blanched extremity.
4. Cuff slowly deflated (approx.6-7 mm/sec.)and pressure at which flush first appears is approx. systolic pressure.

Fig. 4.—Diagram of the flush technique for measuring blood pressure in infants. (From Goldring and Wohltmann, J. Pediat. 40: 285, 1952.)

medical management, for too many infants will improve, then deteriorate rapidly and die. Recently it has been the policy to operate as soon as it is established that serious cardiac failure exists on the basis of coarctation, and the child can be brought into optimum condition.

Operative Findings and Results.—In all 16 infants the coarctation was complicated by patency of the ductus arteriosus. In 10, the narrow segment was located proximal to the ductus (preductal) and in 6 immediately adjacent to the ductus (juxtaductal) (Table I). It was not possible to classify any case as definitely postductal. There was considerable variation in the length of the narrowed segment, the extremes ranging from a short pinched constriction (4 infants) to involvement of the entire transverse arch (2 infants).

In general, the longer coarctations were preductal in position, the shorter segment varieties juxtaductal. The diameter of the ductus was also quite variable, and large and small caliber communications were found in both groups. Three infants in Group 1 had shunts equal in size to the aorta.

TABLE I. COARCTATION IN INFANTS—OPERATIVE FINDINGS
PATENT DUCTUS ARTERIOSUS—16 CASES

	GROUP 1		GROUP 2	
Position	Preductal	10 cases	Juxtaductal	6 cases
Procedure	End-to-end	4	End-to-end	6
	Homograft	1		
	Shunt	1		
	None	4		
Survivors		3		6
Deaths		7		0

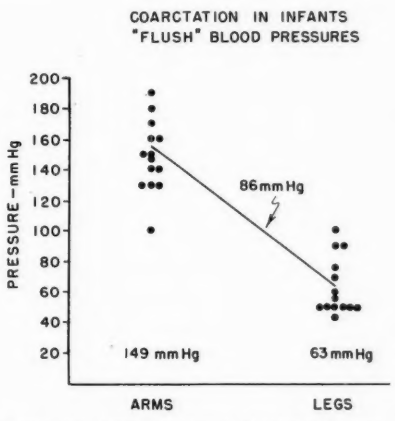

Fig. 5.—This figure shows the blood pressures obtained by the flush technique in 14 of the infants. The gradient across the coarcted segment averaged 86 mm. Hg.

Mobilization and end-to-end reconstruction was possible in 4 cases in Group 1, and in all 6 cases in Group 2. The anastomoses were done with running fine silk sutures, interrupted at frequent intervals. The first child in the series had involvement of the transverse arch, and the defect was bridged by a fresh homograft. The fifth case also involved the arch, and it was felt at the time that a graft could not be properly placed, so the proximal ends of the divided left carotid and the left subclavian arteries were anastomosed. Four infants died during thoracotomy before any definitive procedure could be done.

There were 9 survivors and 7 operative deaths, a mortality rate of 44 per cent. (By comparison, there were 5 deaths in the 72 operated cases over 2 years of age, a mortality rate of 7 per cent; 2 of these were homograft failures.) All 7 deaths were in Group 1 patients and occurred either before, during, or at the end of operation. Cause of death was cardiac arrest in 6, ventricular fibrillation in 1. The mortality rate has declined as experience with these sick children has increased. All 5 infants operated upon during

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the first 5 years died. During the last 5 years there have been 11 cases with only 2 deaths, a mortality rate of 18 per cent for this abbreviated group. These fatalities were in 2 of 4 cases done with hypothermia. Body cooling has not been used since 1956, and there have been no spinal cord sequelae, in spite of rather extensive mobilization of the aorta in several instances.

Follow-Up.—Of the 9 operative survivors, 8 are living and clinically well. One infant, repaired at 3 weeks of age, did better for about 3 months, and then returned with recurrent failure, and signs indicating a severe intracardiac abnormality. It was hoped that he could be improved to the point where open heart surgery would be feasible, but death occurred at 9 months of age. Autopsy revealed a large atrial septal defect of the ostium primum type, with gross deformity of both the mitral and tricuspid valves. The aortic repair was intact, and the diameter measured 12 mm. at the suture line, 17 mm. above and below.

The remaining 8 children have been followed from 9 months to 5 years since operation (average 2 years) (Table II). All are asymptomatic at the present time, although clinically 3 children definitely have an intracardiac left-to-right shunt, and 2 others are suspected of having a small shunt. The remaining 3 do not have additional lesions. Growth and development have followed the normal pattern in all but 1, a child with probable ventricular septal defect, who is exhibiting growth failure.

TABLE II. COARCTATION—OPERATIVE SURVIVORS

Follow-up—2 years	
Symptoms—none	
Additional Left-to-Right Shunts	
None	3
Suspected	2
Definite	3
Average Pressure Gradient	
Preoperative	90 mm. Hg
Postoperative	13 mm. Hg

Preoperatively the arm-leg pressure gradient for the survivor group averaged 90 mm. Hg. This has been reduced by operation to 13 mm. Hg, and in 3 the pressures are equal. The highest individual residual gradient is 50 mm. Hg, reduced from a preoperative value of 100 mm. Hg. Technical repair in this case may not be satisfactory, although the child is now 2 years postoperative, and as yet is manifesting no brachial hypertension.

The electrocardiogram pattern and the cardiac contour have reverted to normal in the 3 cases with no residual defects. In the 2 suspect cases, a suggestion of right ventricular enlargement remains by both electrocardiogram and chest film, although there has been definite improvement over the preoperative state. In the 3 children with definite additional defects, there has been marked improvement in the clinical condition, but the electrocardiograms are unchanged, and the chest roentgenograms look very little different, except for a decrease in pulmonary vascular congestion.

We plan to follow the 5 children with possible additional defects closely for the next few years, and perform diagnostic angiocardiography when indicated. In all probability, at least 3 will require open heart repair. All 8 children will be followed through the growth years to obtain accurate information concerning the problem of relative stenosis at the suture line.

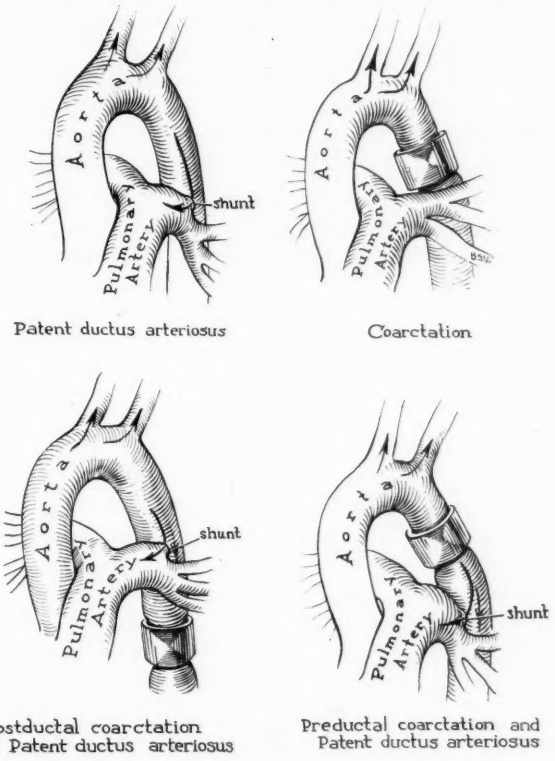

Fig. 6.—Shows the four types of experimental preparations employed in the study of coarctation and patent ductus.

EXPERIMENTAL STUDIES

The hemodynamic alterations that occur with isolated aortic coarctation or patent ductus arteriosus have been amply studied both clinically and experimentally, but investigation of the changes that occur with the two lesions in combination have not been reported. To answer some of the puzzling questions that have come up while caring for these infants, the following long-time experiments were done: appropriate base line data, including carotid and femoral arterial pressures, right heart catheterizations, chest roentgenograms, and electrocardiograms were obtained in a number of dogs, and then four categories of experimental animals were prepared. These

were: (1) patent ductus only—4 dogs, (2) coarctation only—4 dogs, (3) patent ductus and postductal coarctation—8 dogs, and (4) patent ductus and preductal coarctation—7 dogs (Fig. 6). The aorta-pulmonary artery shunts were all of the Potts type, constructed as near 5 mm. in length as possible. Gradually progressive coarctations were produced by the use of cylinders of Ameriod plastic encased in stainless steel jackets, a method described by Berman.[11] Total occlusion of the aorta occurs with this method in 2 or 3 months. Dogs of uniform size and weight were used to facilitate comparison between groups. The animals in each group were then followed, and the base line studies repeated at intervals until death or sacrifice. Ninety-three complete sets of postoperative observations were done in the 23 dogs, an average of four observations per dog. Complete autopsy studies were done by a member of the Pathology Department.

Results.—Group 1 (isolated patent ductus) and Group 2 (isolated coarctation) were used as controls. Each lesion was tolerated exceedingly well (Table III). In Group 1, a loud continuous murmur was present in all 4

TABLE III. FATE OF EXPERIMENTAL ANIMALS

	LIVED	DIED
Patent Ductus	4	0
Coarctation	3	1
Postductal	3	5
Preductal	0	7

dogs, and they remained asymptomatic until sacrificed (average 7 months). The animals in Group 2 likewise remained vigorous and healthy. All developed a pronounced collateral circulation. One dog died of a ruptured aorta at the site of the ring at 50 days, and the remaining 3 were sacrificed, 2 at 5 months and 1 at 15 months. No dog in either group developed roentgenographic or electrocardiographic changes of significance.

The clinical course of the animals in Groups 3 and 4 was quite different. In Group 3 (patent ductus with postductal coarctation) 4 of the 8 dogs developed the signs and symptoms of cardiac failure, with dyspnea, tachycardia, pulmonary congestion, ascites, and edema. Cardiomegaly was present. There was no response to digitalis therapy, and they died 2, 4, 6, and 36 weeks after operation. A fifth animal died of aortic rupture. The other 3 dogs remained asymptomatic, in spite of a functioning ductus and an evident coarctation, until they were sacrificed 1 year postoperatively. In Group 4 (patent ductus and preductal coarctation) the combination of lesions was the least well tolerated. All 7 animals developed severe congestive failure. Survival time varied from 13 days to 56 days (average 39 days). Chest films showed a pronounced degree of cardiomegaly and vascular engorgement. A few animals in Groups 3 and 4 developed questionable right ventricular enlargement by electrocardiogram.

Fig. 7 shows the blood pressure response to the various procedures. Two values are shown, the preoperative and the final observation before death or sacrifice. Although the spread of mean pressures was moderately large,

the directional trends are accurate, for no animal had a pressure response opposite to that depicted for his group. With patent ductus, mean carotid pressure was maintained while mean femoral pressure fell. In isolated coarctation, mean carotid pressure was elevated while the femoral pressure remained

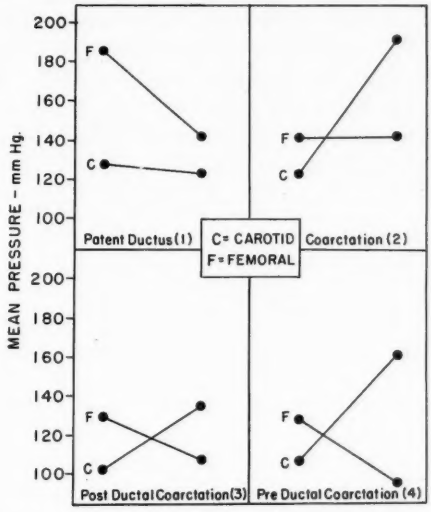

Fig. 7.—Shows the systemic pressure response to the various operative procedures. (Description in text.)

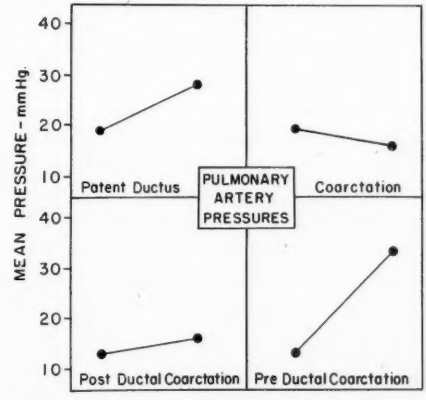

Fig. 8.—Shows the pulmonary artery pressure response to the various operative procedures. (Description in text.)

the same. In Groups 3 and 4, in spite of a different spatial relationship between coarctation and shunt, the over-all response was the same, a rise in mean carotid pressure and a fall in mean femoral pressure. Fig. 8 shows the pulmonary artery pressure depicted in the same way. In Groups 1, 2, and 3 the operative procedure had no significant effect, while in Group 4 (preductal

coarctation) mean pressure was elevated to 33 mm. Hg. Right ventricular diastolic pressures were never significantly above the zero level in any group.

Autopsy examination of animals dying in failure revealed edema, pleural effusion, pulmonary and hepatic congestion. Collateral circulation was most pronounced in isolated coarctation, less so with the combined lesions. Measurements of right and left ventricular wall thickness showed a pronounced degree of left-sided hypertrophy with isolated coarctation, less when a ductus was also present. Right ventricular thickness was essentially the same in all groups, but the dogs in Group 4 had right ventricular dilatation in addition. Study of the lungs revealed small vessel thrombi and intimal proliferative changes to a moderate degree in Groups 3 and 4, but these findings were not pronounced.

DISCUSSION

A significant clinical experience with aortic coarctation complicated by patent ductus arteriosus has convinced us that this combination is a most malignant one, and unless vigorous treatment is instituted almost from birth, the mortality will approach 100 per cent. We are also of the conviction, on the basis of our discouraging experiences with medical therapy alone, that early corrective surgery must be done, even in the face of congestive failure in a critically ill child, if any number are to be saved. Our surgical results will bear improving, 56 per cent survival in 16 cases, but an encouraging upward trend has been seen in the last 5 years, when there have been only 2 deaths in 11 cases, a survival rate of 82 per cent. Early detection of cases and better anesthetic techniques have contributed greatly. For early diagnosis the condition must be kept in mind, for the infants have a variety of nonspecific symptoms. The flush blood pressure technique has been of immense help.

Attempts to separate these infants into various categories on the basis of clinical or laboratory findings had proved futile for us. Some clinicians rely heavily on the electrocardiogram, believing those children with left ventricular enlargement have less serious lesions and can be managed medically, while those with a right-sided pattern have a graver prognosis, a greater probability of additional shunts, and will usually die unless helped surgically.[3, 9] In our experience, the electrocardiogram pattern has not been this reliable. Table IV shows the relationship between the electrocardiogram, ductus position, and associated defects in this series. Right- and left-sided patterns were found with both anatomic variations of ductus position, and with and without additional left-to-right shunts. We believe that selection of cases for operation should be based on the diagnosis of significant coarctation in any infant exhibiting cardiac failure and cardiomegaly, without regard for the electrocardiogram pattern. In virtually all such cases a patent ductus will be present with the coarctation.

Two objections have been raised to the correction of coarctation in infancy. One is that the mortality will be prohibitively high should an intracardiac lesion also be present. We take the opposite view, and feel that the

only plausible method of management is the prompt correction of the coarctation and division of the ductus, making it possible to defer repair of the intracardiac component to a later time. As yet we have no children carried through the second phase of this plan, but there are 3 in the group that will almost surely need further surgical attention. The other objection relates to the development of a relative coarctation at the line of anastomosis with body growth. No absolute data are available as the follow-up in the reported cases is still too short. Moss and his co-workers[12] have recently reported angiographic studies on 5 infants followed 2 to 4 years after repair, and concluded that after proper corrective surgery the aorta should remain functionally patent, regardless of growth. In any event, it is evident that surgery in infancy is lifesaving, and even if re-operation is necessary during adolescence, this is still an equitable price to pay.

TABLE IV. RELATIONSHIP OF DUCTUS POSITION, ECG, AND ASSOCIATED DEFECTS IN 16 CASES OF COARCTATION AND PATENT DUCTUS

CASE NO.	DUCTUS POSITION	ECG PATTERN	ASSOCIATED DEFECTS	OPERATIVE OUTCOME
1	Preductal	RVE	None	Died
2	Preductal	RVE	None	Died
3	Preductal	LVE	None	Died
4	Preductal	RVE	None	Died
5	Preductal	RVE	VSD	Died
6	Juxtaductal	LVE	None	Survived
7	Preductal	LVE	Fibroelastosis	Died
8	Preductal	RVE	Left-to-right shunt	Survived
9	Preductal	RVE	None	Died
10	Juxtaductal	LVE	None	Survived
11	Preductal	RVE	?Left-to-right shunt	Survived
12	Juxtaductal	LVE	?Left-to-right shunt	Survived
13	Juxtaductal	LVE	None	Survived
14	Juxtaductal	LVE	Left-to-right shunt	Survived
15	Preductal	RVE	Ostium primum	Survived*
16	Juxtaductal	RVE	Left-to-right shunt	Survived

*Died 9 months later.

Very little is known about the precise hemodynamic alterations which make coarctation and patent ductus such a serious combination. Gupta,[13] in acute animal experiments, demonstrated that the opening of a ductal shunt distal to a coarctation makes the constriction physiologically more severe. Thus, the femoral pressures and pulse contours with a 65 per cent coarctation and open shunt resemble those of an isolated coarctation of 75 per cent. This is an important concept if it applies to human cases. It is difficult to believe that the alleged lack of development of collateral channels in the preductal group has much to do with the prognosis. Such channels are actually demonstrable at autopsy in many cases.[2] Clinically it is rare to find an infant with differential cyanosis of the lower trunk, indicating maintenance through the ductus by a "systemic" right ventricle. None of our patients had differential cyanosis, and oxygen saturations done in a number showed no difference above and below. Edwards[14] was the first to call attention to the vascular obliterative disease to be found in the lungs of these infants, and others since have commented on the severe pulmonary hypertension that is present when

such measurements can be made.[9, 12] Although the pathogenesis of the hypertension, and the exact relationship between it and the observed vascular changes are not yet worked out, it is most likely that the pulmonary vascular tree plays a major role in influencing the clinical picture of coarctation and patent ductus, just as it does in so many other congenital malformations (Fig. 9).

Our animal observations are pertinent to these problems. Under experimental conditions the combination of coarctation and patent ductus was also poorly tolerated, whereas the isolated lesion caused little or no difficulty. Cardiac decompensation occurred regardless of the relative position of the shunt to the constriction, although it appeared that the preductal arrangement was the more serious one, and in this group pulmonary hypertension

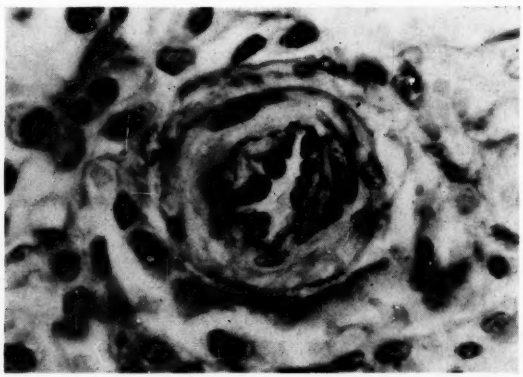

Fig. 9.—Small pulmonary arteriole from an infant with coarctation and patent ductus. The muscular layer is greatly thickened. In comparable vessels from the lungs of normal infants of this age the muscular layer is virtually absent.

was seen. A well-developed collateral circulation did not prevent heart failure. Like its clinical counterpart, the failure responded very poorly to digitalis therapy, and the animals died in spite of vigorous medical treatment. At autopsy, early obliterative changes were seen in the small pulmonary vessels, and, in the preductal group, dilatation of the right ventricle indicated that there was considerable strain on this chamber during life.

SUMMARY

1. Sixteen infants with coarctation of the aorta and patent ductus arteriosus treated by early surgical correction are presented. Clinically all were in refractory cardiac failure, and had marked cardiomegaly. The electrocardiogram pattern was variable. Diagnosis was made by demonstrating a significant pressure gradient by the flush technique.

2. Median age at the time of operation was 6 weeks. Wide anatomic variation in the relationship of the two lesions was encountered. Operative

mortality was 44 per cent for the series. In the last 5 years there have been 11 cases with only 2 deaths.

3. There has been 1 late death. The other 8 children have been followed an average of 2 years. All are asymptomatic. Clinically, several have additional intracardiac shunts which will require closure in the future.

4. We are of the conviction, on the basis of this experience, that prompt correction of the coarctation and division of the ductus is the only rational form of treatment for these seriously ill infants.

5. Observations are presented which demonstrate that coarctation and patent ductus is also a lethal combination in the experimental animal.

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(For Discussion, see page 85.)

PROBLEMS IN THE SURGICAL MANAGEMENT OF COARCTATION OF THE AORTA

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DEFINITIVE resectional surgery for coarctation of the aorta was introduced 15 years ago.[1, 2] During the ensuing years, thousands of patients have been successfully operated upon and the surgical technique has become well standardized. Today, the operative mortality is very low and, in general, the mere diagnosis of coarctation is in itself clear and sufficient indication for surgery. Despite these facts, coarctation of the aorta is not completely understood and retains problematical therapeutic aspects. These problems may appear during the operation or in the postoperative period and, as will be shown in this paper, they usually can be solved without jeopardizing the life of the patient. However, these problems raise questions concerning surgical judgment and the requisites of a satisfactory surgical result. We do not venture to offer definitive solutions to these problems but, rather, in this paper, we present our findings with the object of stimulating worthwhile discussion that ultimately should benefit future patients with coarctation.

SURGICAL EXPERIENCE

Our surgical experience with coarctation to January, 1959, includes 60 patients, all operated upon within the past decade. There have been 7 operative deaths and in 1 patient no definitive operation was accomplished. It will be seen from Fig. 1 that this is a safe operation, if the extremes of age in the life history of the disease are avoided. The two deaths in the middle-age span occurred in (1) the first patient in the series who died of a delayed anastomotic leak, and (2) the only patient in the series in whom it was attempted to swing down the very large subclavian artery for end-to-end anastomosis below the coarctation. The technique worked nicely; however, the patient died suddenly approximately 6 hours after operation. The crotch between the aorta and the subclavian artery had split open where it had been hyperextended. Pathologically, there was found to be a loss of elastic tissue in this region. This experience led us to avoid using this method and certainly nowadays the use of grafts has rendered this technique obsolete.

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Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

The problems that have been encountered in this operative experience may be broadly grouped under five headings: (1) coarctation diagnosed in patients 1 year of age or less; (2) coarctation diagnosed in patients more than 45 years of age; (3) the syndrome of postoperative abdominal pain; (4) the size of the anastomosis; and (5) residual hypertension.

1. *Patients 1 Year of Age or Less.*—Eleven patients 1 year of age or less have been operated upon for coarctation. This seems like a high percentage of our total operative experience and is probably not a true indication of the relative frequency of significant symptoms in this age group. It reflects both an active pediatric cardiological service and pediatricians alerted to the significance of feeling the femoral pulsations. All 11 patients manifested congestive heart failure (Table I).

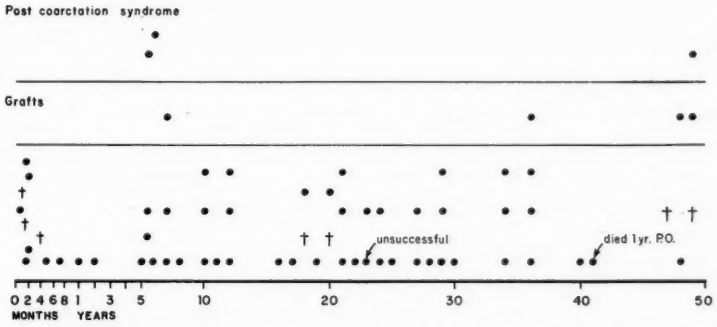


Fig. 1.—Summary of the operative experience upon which this paper is based. The ages of all 60 patients are indicated at the bottom of the figure. Details of the 7 operative mortalities are discussed in the text. The patient designated "unsuccessful" had an aneurysm distal to the coarctation. This was early in our experience, grafts were not available, and no definitive operation was accomplished. The patient who died 1 year following surgery died of complications of rheumatic heart disease. All of the other patients are known to be living. The middle section of the chart shows that grafts were used in only 4 of the 60 patients. The postcoarctation abdominal pain syndrome was recognized on only 3 occasions.

An important question that remains to be definitely settled regarding the advisability of this operation on infants is whether they will "outgrow" their anastomoses and relative coarctation will recur. The precaution of using interrupted suture technique should be followed because such anastomoses have grown well experimentally, but the possibility of the recurrence of coarctation is an important theoretical objection to performing this operation electively on infants.[3, 4] In this regard, our longest postoperative follow-up is only 4 years and there is no clinical evidence of recurrent coarctation at the present time. Others undoubtedly have additional evidence on this point.

In a recent paper, Moss and associates,[5] on the basis of five postoperative angiographic studies, suggest that growth of anastomoses lags relative to normal infantile aortas. However, in our experience many of these aortas are definitely abnormal (hypoplastic). Furthermore, it may be technically impossible in minute aortas to perform an "ideal" anastomosis that leaves

TABLE I. TABULAR SUMMARY OF PERTINENT DATA ON 11 PATIENTS OPERATED UPON 1 YEAR OF AGE OR YOUNGER

NAME	AGE	SEX	CLINICAL ASPECTS	CATHETERIZATION FINDINGS	OPERATIVE FINDINGS	POSTOPERATIVE COURSE	COMMENT
K. O.	3 wk.	M	Cyanosis, tachycardia, cong. fail., and Wolff-Parkinson-White syndrome	Not catheterized	Typical + PDA, cardiac arrest	Benign; good femoral pulses	
S. S.	5 wk.	M	Cyanosis, cardiomegaly, cong. fail.	Coarctation, IASD, ? PDA	Typical, small PDA	Benign; subsequent oper. for mediastinal mass (hyperplastic thymus)	Postop. catheterization, femoral artery pressure rose from 45/37 to 110/70
P. L.	5 wk.	F	Dyspnea, cyanosis, cong. fail.	Coarctation, PDA, IVSD	Hypoplastic arch, big PDA, systemic PA pressure did not fall on dividing PDA	Resp. failure and cardiac arrest 2nd postop. day, resuscitated; died 3 wk. postop.	Autopsy; Eisenmenger
D. N.	6 wk.	F	Cyanosis, tachypnea, cong. fail.	Coarctation, ? lt.-to-rt. shunt	Atresia of aortic arch, PDA supplied descending aorta	Died after surgery	Autopsy; atresia from innominate to subclavian, subaortic stenosis, IVSD, PDA
D. W.	7 wk.	M	Cyanosis, dyspnea, cong. fail.	High coarctation with hypoplasia of arch	Coarctation at subclavian wedge resection	Benign; progressive decrease in heart size	
K. Y.	7 wk.	M	Dyspnea, cong. fail.	Coarctation, ? myocardial disease	Typical, hypoplastic arch	Benign; decreased heart size	
B. W.	2 mo.	F	Cardiomegaly, cong. fail.	Coarctation, hypoplastic arch, IVSD	Typical, much collateral, distal arch hypoplastic	Benign; relative narrowing at anastomosis	Postop. catheterization, femoral pressure rose from 70/46 to 102/60; no residual shunt detected
J. A.	4 mo.	M	Dyspnea, cong. fail.	Coarctation, IVSD, pulm. hypertension	Small PDA, hypoplastic aorta, coarctation not recognized	Died postop.	Autopsy; coarc. in hypoplastic segment, IVSD
M. E.	5 mo.	M	Grand mal, cong. fail.	Coarctation, PDA above intracardiac lt.-to-rt. shunt	Typical	Benign; IVSD closed 6 mo. later	Femoral pressure: preop. 64/72—postop. 98/56
N. G.	7 mo.	F	Tachypnea	PDA, IVSD (coarctation missed)	Large PDA, very small proximal aorta	Benign; heart size ⅓ smaller	Postop. catheterization, rt. vent. pressure fell from 89/3 to 40/7; residual narrowing in hypoplastic segment
W. W.	1 yr.	M	Poor development, pulm. congestion, cardiomegaly	PDA (coarctation missed)	PDA, coarctation below diaphragm excised, Heineke-Mikulicz repair	Benign; heart smaller	

PDA = patent ductus arteriosus; IVSD = interventricular septal defect; IASD = interatrial septal defect.

no local deformity. Under such circumstances, normal findings on postoperative studies must be exceptional. Fortunately it is function and not appearance that counts and none of their re-studied patients had residual pressure gradients.

With the recent rapid development in pediatric cardiology, it has become apparent that coarctation is a significant cause of left heart failure in infancy. It is our current belief that, if these infants do not respond promptly and completely to anticongestive measures, surgery offers less risk than protracted medical treatment. In the accompanying chart (Fig. 2) there is evidence that these infants get into difficulty because of associated additional congenital cardiac defects. Seven of the 11 infants were found to have intracardiac left-to-right shunts; 2 others had only extracardiac shunts (patent ductus);

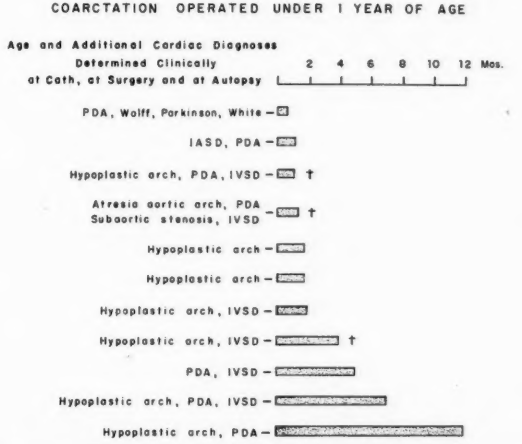


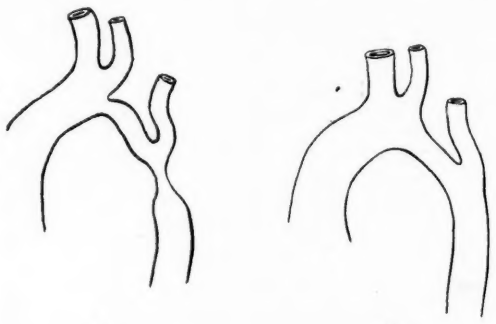
Fig. 2.—This figure emphasizes the large number of associated cardiac defects in infants having symptomatic coarctation. Seven of the 11 patients had associated intracardiac shunts and 2 additional ones had patent ductus arteriosus. In all, there were 7 patent ductus. In 8 of the 11 cases there was hypoplasia or atresia of the aortic arch system. The 3 patients who died as a result of operation had palpably systemic pulmonary hypertension, and, in all 3, including the 2 with patent ductus, this pressure did not fall as a result of the operative procedure.

each of the 3 patients who did not survive surgery had a large interventricular septal defect with systemic pulmonary hypertension. These findings indicate that the coarctation may be the straw that breaks the camel's back. Hopefully, correction of the coarctation may be lifesaving and allow future correction of the other anomalies. Subsequent studies on 2 survivors with intracardiac shunts revealed dramatic improvement. One lost all catheterization evidence of an interventricular septal defect. This suggests that a fall in left ventricular pressure may allow a small shunt to close. In a second patient, who also had a large patent ductus immediately above the coarctation as well as an interventricular septal defect, the right ventricular pressure fell from 89/3 to 40/7 mm. Hg. This relationship of coarctation and ductus may result in extreme pulmonary hypertension. A third infant, 5 months old at the time

PDA = patent ductus arteriosus; IVSD = interventricular septal defect; IASD = interatrial septal defect.

of division of his coarctation, retained systemic pulmonary hypertension due to an interventricular septal defect. This was closed by open cardiotomy 6 months later, and he has done well since.

In the adult coarctation there often is hypoplasia of the proximal aortic segment. This is also seen in the infant in whom this segment may seem extremely small (Figs. 3, 4, 5). Under these circumstances, in 2 infants it was impossible from the outside of the aorta to suspect that, in addition, there was a diaphragmatic obstruction typical of coarctation. In the first of these patients, a patent ductus was divided and, although coarctation was suspected from contrast studies at catheterization, no aortic resection was done and the coarctation was proved at necropsy on the following day. On the basis of this experience, the second case was recognized and treated properly. Frequently the coarctation deformity of infancy is less obvious than that seen

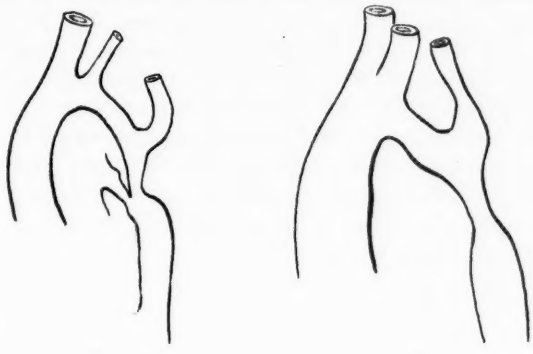


A. *B.*

Fig. 3.—Tracings of enlargements of single frames of 35 mm. motion-picture film on 2-month-old infant B. W. *A* depicts the preoperative status. *B* is from a selective angiocardiogram done 1 year postoperatively. On the basis of the extremely hypoplastic distal arch depicted in *A*, it was suggested that this patient might well prove to have a surgically hopeless problem. Note that there is no residual anastomotic narrowing, but that a definite relative hypoplasia in the distal aortic arch persists. The femoral artery pressure to direct measurement at the time of the studies (*A*) was 70/46; at the time of the studies (*B*) it was 102/60.

later in life. It is likely that the coarctation represents a point of fixed narrowing and with growth the adjacent aorta enlarges to give the typical hourglass deformity. It is suggested that when in doubt the suspected segment of aorta should be longitudinally incised so that, if a coarctation is not found, the aorta can be reconstituted. However, each time we have done this, the longitudinally incised segment has been excised as a part of the coarctation specimen with the exception of 1 patient in whom it was believed wise, because of the small caliber of the vessels involved, to treat the coarctation by closing the incision in Heineke-Mikulicz fashion after excising the diaphragm. The only other unorthodox anastomotic procedure was in another extremely hypoplastic aorta in which the anterior two thirds of the coarcted segment was excised as a transverse wedge and an end-to-end anastomosis was done in the usual fashion, except that the posterior portion of the aorta remained intact.

The infant who has a hypoplastic proximal segment, be it a long portion of the arch or only the segment from the subclavian to the coarctation, will, of course, have an anastomotic lumen no larger than this segment. This may be only one fourth or one third the diameter of the descending aorta beyond the anastomosis. We have several postoperative studies which suggest that



A. *B.*

Fig. 4.—Tracings of enlarged single frames of 35 mm. motion-picture film from infant S. S. *A* is a preoperative one, when the infant was 5 weeks old. *B* is from studies 7½ months later. At the time of surgery, in addition to the patent ductus, there was a diaphragmatic component to the coarctation situated at the narrowest point shown. This could not be seen on the angiographic studies due to insufficient contrast. Note that in the postoperative study the relative hypoplasia of the proximal aorta persists essentially unchanged. Directly measured femoral artery pressures at the time of these two studies were 45/37 and 110/70, respectively.

Fig. 5.—Postoperative angiographic study traced from an enlargement of a 35 mm. motion-picture frame on Patient N. G. The child was 2 years of age at this time and the study was made 16 months after surgery. At the time of surgery, the operator commented upon the very small size of the proximal aorta. There was also a very large patent ductus arteriosus. Certainly the relative hypoplasia has persisted. Residual anastomotic deformity of this degree is probably not hemodynamically significant, but this child should certainly be restudied in several years to see if the deformity increases with growth.

this size discrepancy persists (see Figs. 3, 4, 5). Application of Bernoulli's principle to such a system reveals that the pressures are such that the narrowing may conceivably be self perpetuating (Fig. 6).[6, 7] Nevertheless, the 3 surviving patients in whom this size discrepancy was most marked have done well, recovered from heart failure, and do not need digitalis.

The anatomy of coarctation in the infant is highly variable. However, the only striking difference between infants and adults lies in the group of patients with long segments of narrowing, or even atresia, of the aortic arch. These patients with so-called "infantile" types of coarctation do not often survive to be seen as adults. We ordinarily obtain angiographic demonstration of coarctations only in exceptional cases; particularly in the adult group with grafts readily available. However, all but 1 of the 11 infants were catheterized and, in most, selective angiocardiograms also were obtained. In view of the high incidence of associated defects we now believe such study usually is indicated. But too much reliance should not be placed on unfavorable angiographic pictures of the aortic arch, and almost without exception the infant with coarctation in heart failure should be explored. Although the use of grafts does not seem practical, 2 patients survived operation who were believed to have inoperable conditions on the basis of angiographic studies; the coarctations were not as proximal or as long as predicted.

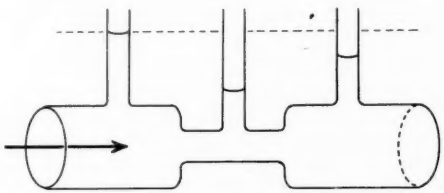


Fig. 6.—Most readers will recall a comparable drawing in their textbook of high school physics. Stated in its simplest terms, the principle is that in any fluid system greater speed of flow is associated with lower pressure. In the tubing depicted above with a constant flow of fluid going from left-to-right it is obvious that the fluid travels faster through the "coarcted segment." The pressures are measured in the manometers taking off from the various portions of the tubing. A more refined example of Bernoulli's principle is the airplane wing in which case air has to travel farther to pass over the curved upper surface than the flat bottom surface; therefore speed is greater, and pressure lower above the wing, and the wing thus acquires lift. If, in the system depicted, the narrowed segment is made out of soft rubber, one will find that as the flow is increased the soft segment can be made to collapse together, indicating a pressure lower than the surrounding atmospheric pressure.

2. *Coarctation in Patients More Than 45 Years of Age.*—The commonest causes of death in patients with untreated coarctation of the aorta are complications of degenerative vascular disease, either closely related to the coarctation itself or in the cerebral circulation. Obviously the tendency to premature development of arteriosclerotic and other degenerative vascular changes increases the operative risk with advancing years, but it is not rational to set a hard and fast age deadline beyond which coarctations should not be resected. The 3 oldest patients in this series were 47, 48, and 49 years of age, respectively. The 47-year-old patient had an uneventful operation, although the aorta was extremely friable and held sutures poorly. She was readmitted to the hospital 3 weeks following operation with fever, dysphagia, and hoarseness, and died shortly after this with a mycotic false aneurysm at the anastomotic site. The 48-year-old patient required a graft for anatomic

rather than degenerative reasons and has had an uneventful postoperative course for more than 1 year. The 49-year-old patient presents an interesting problem and will be briefly discussed.

A 49-year-old white man had been aware of high blood pressure since the age of 11 years. At the age of 36 years, 13 years prior to examination here, he had a cerebral vascular accident with transient left hemiplegia. At this time the diagnosis of coarctation was established. In the interim, the patient had led a moderately sheltered existence and stated that his blood pressure had ranged from 230/120 to 260/120 mm. Hg. Within the 3 months prior to being examined here, the patient had noted the onset of progressive dyspnea, paroxysmal nocturnal dyspnea, and a nonproductive cough. During this time he developed auricular fibrillation and was digitalized. Hospitalization was precipitated by the progression of his symptoms in spite of treatment. Under hospital conditions it was not difficult to control his congestive failure. Blood pressure was reported at from 250/80 to 290/110 mm. Hg in the arms. No blood pressure and only equivocal pulses could be detected in the legs. It seemed clear that this patient had little future without surgery and the patient elected to undergo operation after frank discussion.

The coarcted segment was resected. A 5 cm. homograft was used chiefly to get beyond atherosclerotic changes that were maximal in the immediate vicinity of the coarctation. The operative procedure was uneventful. Early in the postoperative period the blood pressure in the upper extremities fell to approximately 140/90 mm. Hg (Fig. 7). At this blood pressure, the patient had a rapid, weak pulse, was cold to the touch, and covered with profuse perspiration. He presented the typical picture of deep surgical shock in spite of a "normal" blood pressure. His condition did not respond adequately to blood transfusion and vasopressor agents. On the first postoperative day he was placed in a "G-suit"[8] which compressed his body from the waist down. The systolic blood pressure was easily maintained at approximately 180. The patient lost his appearance of shock and felt well subjectively. During the next 16 hours, whenever the "G-suit" was deflated, the patient's pressure fell and the shock-like state recurred. Four days postoperatively the clinical picture progressively improved and the blood pressure stabilized at 190/90 in the upper extremities with a pressure in the legs of 175/80. Six days postoperatively, the picture became complicated by abdominal pain and distention of an ill-defined nature and mental confusion. This confusion and mildly progressive abdominal symptoms and signs continued until death in coma occurred on the eighth postoperative day.

Necropsy showed severe arteriosclerosis in the remaining aorta and in both the cerebral and coronary arteries. There was extensive recent encephalomalacia in the right cerebral hemisphere. Also noted were three berry aneurysms in the middle cerebral arteries. An additional finding which will be discussed subsequently was a necrotizing angiitis of the "acute, hypersensitivity type" that was evident in colon, renal arteries, and prostate.

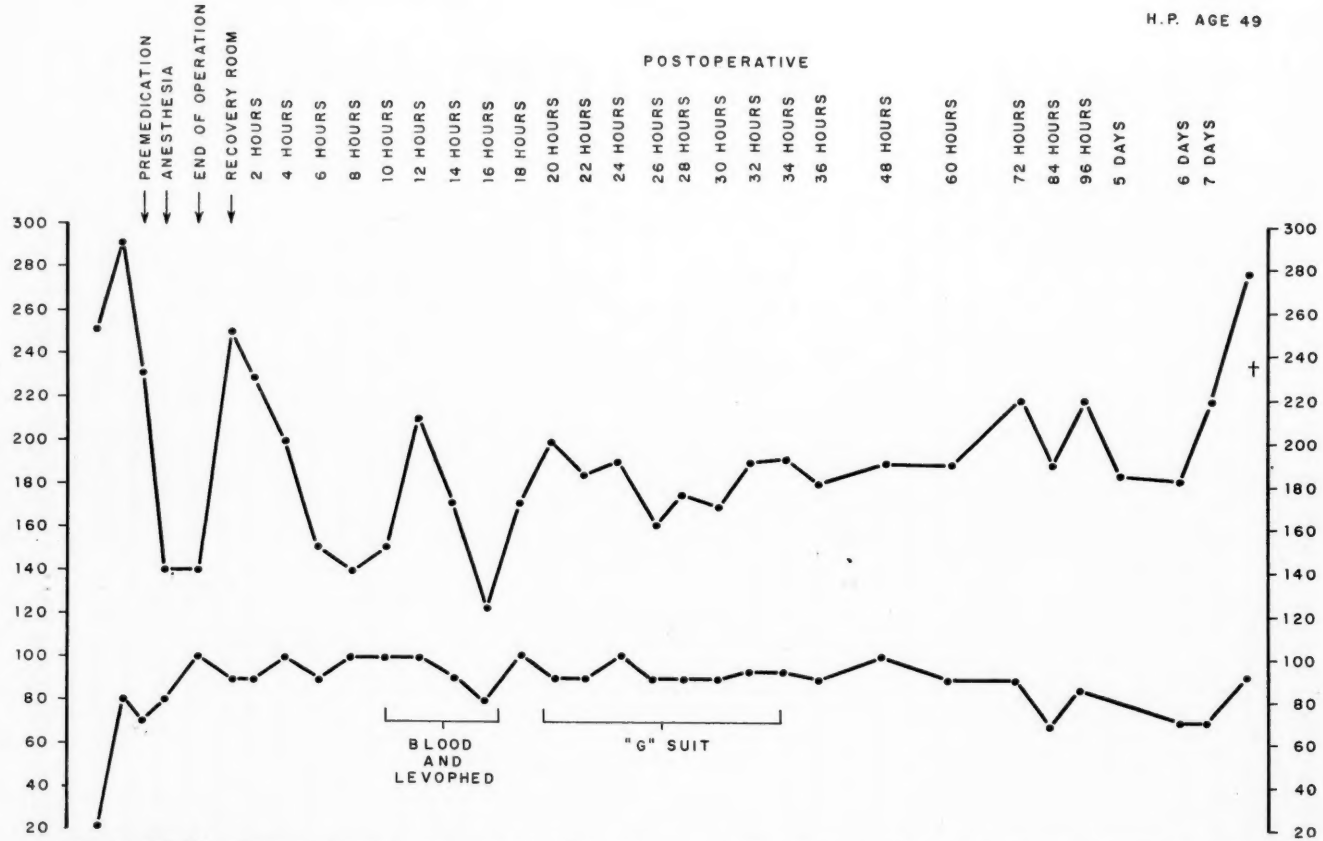


Fig. 7.—Graphic summary of systolic and diastolic blood pressure in Patient H. B., aged 49 years. Note that there was no stability to his postoperative blood pressure until the G-suit was applied, approximately 16 hours after surgery. During the period depicted, the clinical course and necropsy findings proved that the circulation maintained was inadequate to perfuse large portions of the brain.

In brief, this man died as a direct result of inadequate cerebral perfusion at the lowered postoperative blood pressure. No discrete vascular occlusions could be demonstrated in the cerebral circulation. The only finding was advanced arteriosclerotic changes with resultant severe narrowing. Paradoxically the situation resulted in which the cerebral circulation would not tolerate the lowering of blood pressure associated with a technically satisfactory operation. Admittedly this case is an isolated occurrence in our series. However, it is a readily explainable complication and theoretically may occur with some frequency in patients of this age group. We should be interested to know if others have encountered similar problems.

3. *Syndrome of Postoperative Abdominal Pain.*—The syndrome of abdominal pain following surgical resection of coarctation occurs with sufficient frequency so that most thoracic surgeons have observed it. Sealy[9] first mentioned such a case in 1953 and described what has proved to be the typical pathologic picture. Other case reports followed,[10, 11] but it remained for Ring and Lewis[12] to assemble a group of these cases and forcefully bring to the attention of thoracic surgeons the true significance of this problem. A group of poorly understood and presumably coincidental postoperative abdominal complications suddenly made sense and were obviously thereafter better cared for.

The incidence of this problem may be surprisingly high. Ring and Lewis[12] report some degree of the syndrome in 28 per cent of 64 postoperative patients. It is difficult to draw the line between more than the usual postoperative distention and ileus, and a true abdominal complication. A review of our records revealed only 4 clear-cut examples of worrisome postoperative abdominal problems in 60 cases, and 1 of these proved to be an unrelated condition.

It seems clear that the basic pathologic lesion is a mechanically induced panarteritis in the visceral arterial tree. The mechanical insult is related to the increased pressure head and pulsation resulting from relief of the obstruction in the aorta. Comparable vascular lesions have been experimentally produced in the kidneys of rats, in which animals the splanchnic circulation is apparently relatively more resistant.[13] The renal arteries of the 49-year-old man cited above did show such changes at necropsy. Renal arterial changes may well be common after operation for coarctation, but do not manifest themselves as overtly as do changes in the mesenteric circulation. Sealy and associates[14] state that their experience suggests that the abdominal pain syndrome occurs in those patients who have a reactive hypertension of greater than preoperative levels occurring after the second postoperative day, and lasting for from 1 to 2 weeks. In a few patients, we have observed and have been quite concerned about this type of reactive hypertension; however, these individuals have not shown the abdominal pain syndrome.* It is possible,

*In a 5-month-old infant operated on too recently to be included in this series, the syndrome appeared in the presence of definite reactive hypertension (blood pressure up to 150/100 mm. Hg). On Apresoline therapy, the course was uneventful.

Fig. 8.—Example of reactive postoperative hypertension seen in a 23-year-old woman, V. G. Note that her preoperative blood pressure was quite labile; however, prior to surgery it had subsided to approximately 150/90. Note that 9 weeks were required after surgery before the pressure has returned essentially to these levels. This picture is common after surgery for coarctation of the aorta; however, except for the infant mentioned in the footnote, we have not recognized the abdominal pain syndrome associated with it. The hypertension causes the surgeon concern about the anastomosis, but to date such concern has been unfounded in our experience.

however, that reactive hypertension is related to acute postoperative renal vascular changes (Fig. 8). Two children who had the syndrome of postoperative abdominal pain in its classic form had no dramatic fall in blood pressure in the immediately postoperative period, but, in each, the systolic pressure was essentially unchanged from the preoperative level during the first postoperative week. In the third patient, the 49-year-old man already reported, the fall in blood pressure was immediate and rather dramatic. However, it has seemed to us that in all three of these instances a complete relief of obstruction was accomplished with absolutely no anastomotic narrowing or associated aortic hypoplasia and thus the mesenteric arterial tree received maximal increase in blood flow. This, of course, is a common operative accomplishment.

One of the 2 children with the typical syndrome was a 6-year-old boy who had uneventful resection of a typical coarctation. He first complained of abdominal pain on the third postoperative day. At that time the diagnosis of intussusception was considered; a barium-enema study was performed and the examiner thought that he saw and reduced an intussusception. However, the same clinical picture of abdominal pain of fluctuating severity with a paroxysmal component persisted. There were no true localizing signs. The leukocyte count approximated 20,000 per cu. mm. On the eighth postoperative day, a rather severe exacerbation of abdominal pain with some localization to the right side occurred and a laparotomy was performed. The significant findings were injection of the mesenteric vessels in the mid-portion of the small intestine with mesenteric lymphadenopathy. There was a small amount of soapy fluid within the abdominal cavity. Biopsy of the mesentery and a mesenteric lymph node, not including significant blood vessels, showed chronic inflammation and fibrosis. The lymph node showed chronic lymphadenitis. Following laparotomy, the patient's condition remained unchanged for 1 additional week after which his symptoms progressively disappeared, undoubtedly in spite of, rather than because of, the nonspecific therapy he had been receiving. There have been no difficulties in the ensuing 3 years.

The other child was a 5½-year-old boy. At catheterization, a patent ductus and a coarctation with moderate pulmonary hypertension were diagnosed, and at operation the diagnoses were confirmed. The patent ductus was of moderate size and straddled the point of maximum coarctation. The operative procedure was uneventful, and he first complained of ill-defined abdominal pain on the fifth postoperative day. By this time we were familiar with the syndrome and followed him closely. There were several other interesting manifestations, including a migrating arthralgia of the lower extremities, and a peculiar deep swelling in the left lumbar muscles. The child was treated with steroids which relieved the arthralgia, but the abdominal component continued with a fluctuating severity and by the twelfth postoperative day definite ascites was present. There was finally sufficient improvement to allow hospital discharge 1 month after surgery. He was examined again 2

months after operation when it was apparent that he was still having abdominal difficulties; the condition at this time resembled partial intestinal obstruction. He was hospitalized and barium studies confirmed the presence of partial small-bowel obstruction for which reason laparotomy was performed. The terminal 2 feet of ileum was involved in an inflammatory mass and upon taking this down there were multiple fistulas between the loops. A small abscess was also present. This bowel was resected and an anastomosis performed. The patient had an uneventful convalescence and pathologic examination of the resected bowel showed ulcerations, fistulas, and a small segment of complete dissolution of continuity of the small bowel. Examination of arteries in the specimen showed only rare, slight intimal proliferation. There was no necrotizing vascular lesion. However, this operation was performed more than 2 months after the initial procedure and presumably the operative specimen represented the end stage of this pathologic process with localized bowel infarction.

The fourth abdominal problem mentioned, which was unrelated to the complication under discussion, occurred in a 7-year-old boy. He had undergone resection and graft replacement of a coarctation and a distal aneurysm. Infected thrombi were present in the aneurysm. After several days of abdominal pain, he required emergency laparotomy for an abdominal catastrophe and associated shock. A spleen, containing multiple infarcts, was found to have perforated spontaneously. This case is cited to warn against overconfidence in expectantly treating such patients. In general, however, the syndrome of abdominal pain is a nonsurgical problem.

Sealy[14] has reported that autonomic blocking agents (hydralazine hydrochloride) are effective. We have not had experience with these agents. One of our patients was treated with steroids with equivocal results, the rationale being that a similar pathologic condition on a hypersensitivity basis may respond to this therapy. It should not be forgotten that these patients may undergo visceral infarction and perforation, and should thus be kept under close surgical scruntiny. Additional reports on specific medical therapy for the underlying vascular lesions should be presented. It is rational that antihypertensive drugs offer specific therapy.

4. *The Size of the Anastomosis.*—The goal in any coarctation operation is resection of the narrowed area and reconstitution of the aorta so that there is a smooth channel without residual narrowing. Unfortunately in many instances this is easier said than done. The typical pathologic feature in coarctation is a rather abrupt narrowing with a definite diaphragmatic component that will usually allow an anastomosis having normal or very nearly normal lumen. However, there are two groups of patients in whom this may not be the case: those having definite hypoplasia of the aorta, and those having a lengthy coarcted segment.

Specific mention of hypoplasia was made in the operative notes of 20 of the 60 patients in this series. The commonest manifestation is the finding

of a small aorta above the coarcted segment, and of some degree of poststenotic dilatation, at least relative to the hypoplastic segment, below the coarcted segment (Fig. 9). The small aorta may constitute only the segment below the left subclavian artery or it may include the whole visible arch. On occasion the entire thoracic aorta may seem definitely small. In either case obviously the anastomosis can be no larger than the diameter of the upper segment and certainly little is to be gained by accomplishing an anastomosis that is larger than the diameter of the distal aortic arch.

Patients having a lengthy coarcted segment are rare. The segment usually is seen as a long tapering constriction; however, in our experience, invariably at the narrow point there is an additional diaphragmatic component. Here one may be obligated to use a graft, but how much less than a normal aortic lumen is acceptable under these circumstances? It has been our practice to try

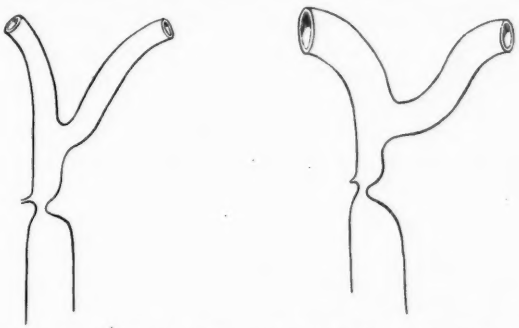


Fig. 9.—Drawings of hypothetical examples of hypoplasia in the aortic arch system. Both of these types are encountered with some regularity. There can be little justification to performing a grafting procedure with a situation as depicted in *A*. An argument can be made in favor of placing a graft from the subclavian artery down to the lower segment in *B*. In such circumstances, we have to date been able to accomplish an anastomosis with a lumen of at least 1 cm. in diameter without resorting to graft.

to accomplish the largest anastomosis possible without the use of a graft. The reasoning behind this choice has been that, in general, these operations are being carried out on young persons with a life expectancy of many years. Since we do not now know the ultimate fate of blood vessel grafts, we have tried to avoid their use when possible. This explains the paucity of grafts in this series where they have been used for degenerative or aneurysmal changes associated with the coarctation (Fig. 10). Arguments can be made against this approach to the problem. These include the possibility of future degenerative changes secondary to the turbulence associated with residual narrowing. Also, Rodbard[7] has shown that localized marked reduction in cross-section area of a vessel may be followed by intimal proliferation with development of a coarctation-like stenosis. He also points out that, in accordance with Bernoulli's principle, the fall in pressure at a point of constriction may well tend to potentiate or increase the narrowing. These changes, however, probably require a more marked and abrupt reduction in cross-sectional

area than would ever be accepted as a satisfactory anastomotic procedure. It is almost an invariable finding that the lumen, through a coarctation of sufficient significance to be diagnosed clinically, is minute. Thus, if a large number of collaterals are not sacrificed (and collateral arteries, usually inter-

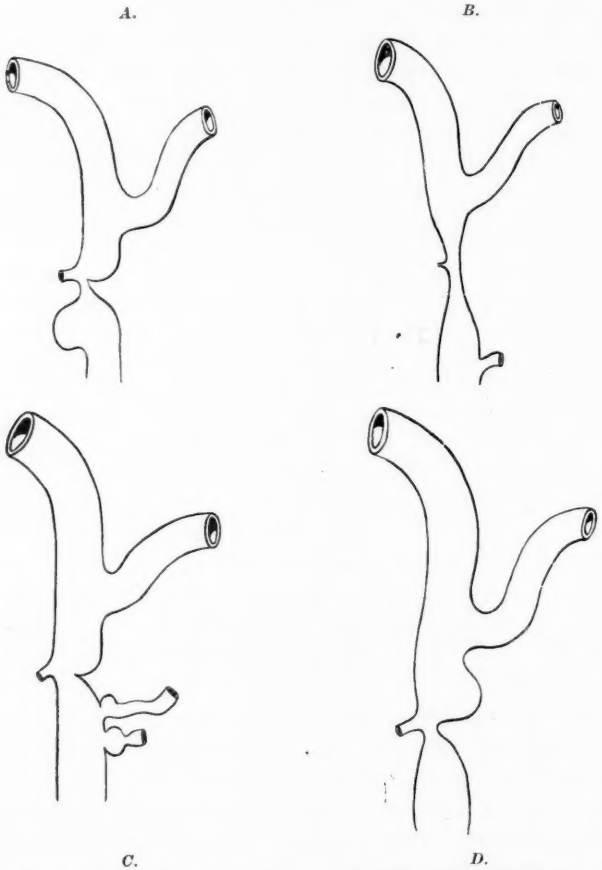


Fig. 10.—Examples of types of coarctation where grafting procedures are frequently indicated. Occasionally the orifices of aneurysmal intercostal vessels, as depicted in *C*, may be carefully purse-string sutured and the involved aortic segment thus preserved. An example of a coarctation similar to *B* is discussed at some length in the text. We would not use a graft in this instance if sufficient narrowed aorta could be resected so that a lumen diameter of 1 cm. could be accomplished.

costals, should not be needlessly sacrificed in accomplishing the operative procedure), the patient should benefit significantly by any increase in diameter that can be achieved.

Gupta and Wiggers,[15] in acute dog experiments, found that a reduction of more than 45 to 55 per cent in aortic cross-sectional area is required before

the pressure in the proximal aorta or femoral artery is affected. This corresponds to a reduction in diameter of one fourth to one third. They also found that total peripheral resistance was not significantly raised until the constriction approximated 70 per cent of the natural aortic diameter. Similarly, Clatworthy and associates[16] found in the dog "little influence on the passage of blood through a stenotic area until that area is reduced to at least 25 per cent of the normal cross-sectional area or about 50 per cent of the normal intraluminal diameter."

A person with coarctation has already made excellent physiologic and anatomic adjustment to much more severe obstruction than that of these experimental conditions, and should thus have normal findings with even greater deformity than obtained in the experimental situations, which is certainly greater than would be acceptable as an operative result. (See the appendix for an elementary mathematical analysis of this problem.)

Data will be presented suggesting that moderate residual constriction is well tolerated by the patient.

In several infants with extremely small aortic segments in the vicinity of coarctation it was tempting to use a graft to enlarge a soda-straw lumen and to overcome the theoretical objection that, in accordance with Bernoulli's principle, these segments may not grow. However, this latter point is not now definitely settled and certainly the use of a static nongrowing graft in an infantile aorta is out of the question. In adults or in large children, with one exception, we have always been able to accomplish an anastomosis, the internal diameter of which approximated at least 1 cm. This is indeed as large as the lumen of many of the hypoplastic aortas and in all cases has given what seems to be a satisfactory operative result without significant residual pressure gradient. An occasional patient has had residual hypertension and we have tried to evaluate the possibility of residual coarctation as an etiologic factor.

5. *Residual Hypertension.*—In the course of postoperative follow-up studies of our patients treated for coarctation of the aorta, it has become apparent that as a group they tend to have extremely labile blood pressures; a reason for this will be suggested. But, in addition to having labile blood pressures, approximately 10 per cent of the patients on repeated follow-up visits remained consistently hypertensive. With the assistance of our Division of Research and Cardiovascular Laboratory,* we have hospitalized 4 of these patients for study, including direct pressure measurements and contrast-medium visualization of the aortas. To throw additional light on this problem, we also have been fortunate in having the opportunity to restudy the 1 patient, mentioned previously, in whom an anastomosis of definitely less than 1 cm. in diameter was accomplished.

She was a 12-year-old child (M. D.), who was asymptomatic, but had typical coarctation findings with blood pressures in the arms of 160/85 to 165/85

*We are indebted to Dr. Harriet P. Dustan, Dr. F. Mason Sones, Jr., Dr. Earl K. Shirey, and Mr. Frederick Olmsted for their assistance.

mm. Hg. Pressures were not obtainable in the legs. The surgical problem and solution as diagrammatically drawn in the patient's chart postoperatively by a member of the house staff are shown in Fig. 11. The reasoning at the time was that, although homografts were readily available, this child's aorta was not completely full grown and the graft might become a point of relative

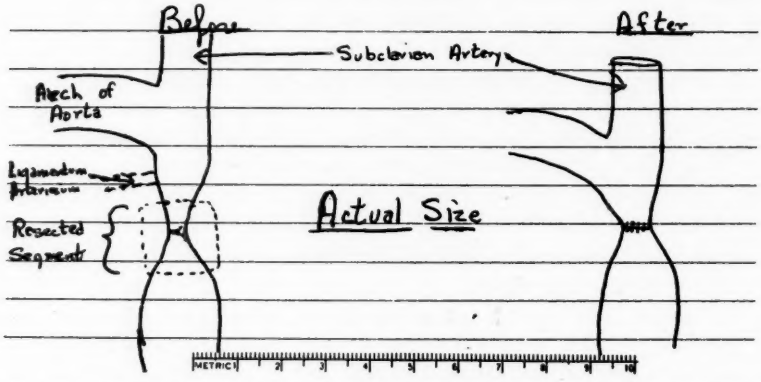


Fig. 11.—Drawing from the chart of Patient M. D. Note that the artist stated the drawing to be actual size and a scale has been affixed so that the reproduced size may be estimated.

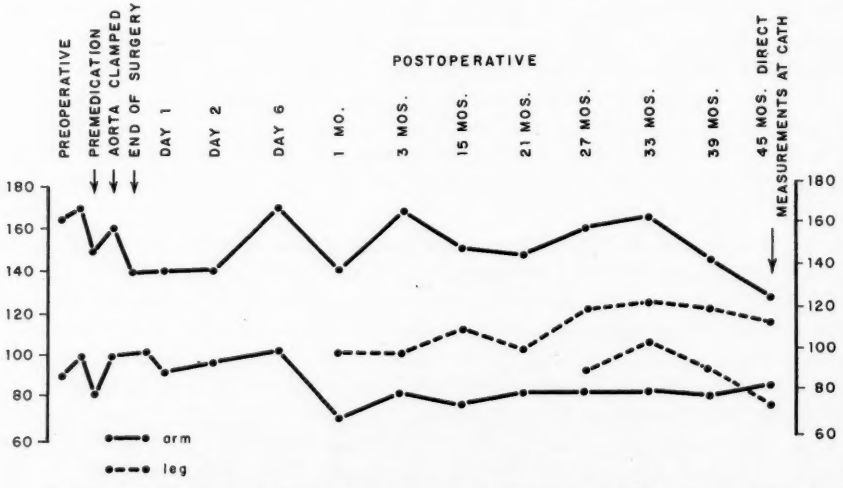


Fig. 12.—Blood pressure observations over almost 4 years on Patient M. D. The decrease in pressure gradients between the arms and legs suggests some aortic growth in the vicinity of the anastomosis. In view of the hypoplasia in her aortic arch system, the residual minor hypertension is probably due to a small central reservoir.

coarctation. More important was concern as to the long-term fate of a homograft placed in a 12-year-old child. This girl has now been observed for 4 years and representative blood pressure readings are shown in the accompanying graph (Fig. 12). Clinically she has remained asymptomatic, although, to palpation, pulses in the lower extremities are not as forceful as

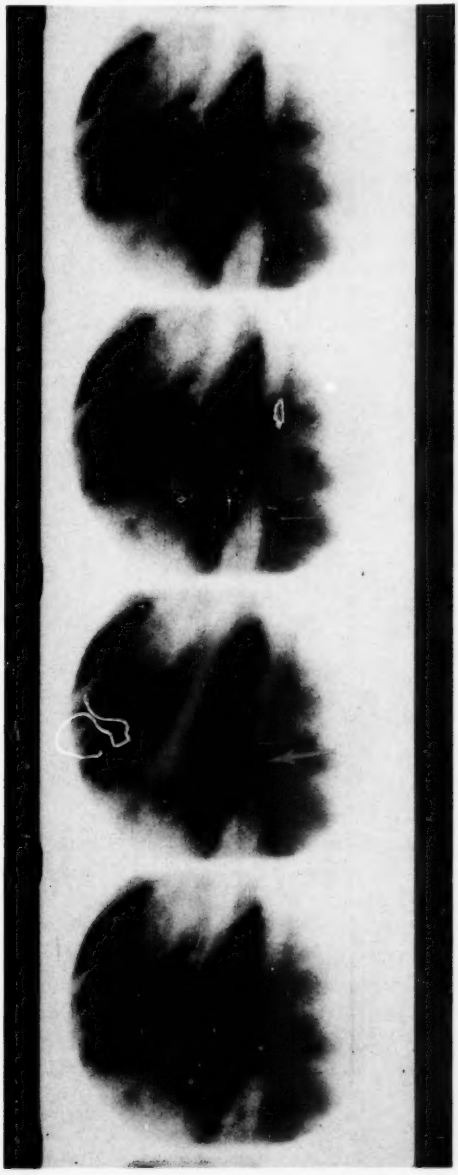


Fig. 13.—Enlargement of 4 frames of 16 mm. motion-picture film which shows injection of contrast medium into the aortic arch of Patient M. D., 4 years after surgery. The next to the bottom frame has been retouched to increase the contrast. The catheter tip can be seen in the innominate artery on the left. Note the small caliber of the transverse aortic arch which is equally as small as the hourglass deformity (*arrow*) of the anastomotic site.

TABLE II. DATA ON PATIENTS STUDIED WITH RESIDUAL HYPERTENSION

PT.	AGE	SEX	PREOP. B.P. ARM	PREOP. B.P. LEG	OPERATIVE FINDINGS	POSTOP. INTERVAL (YR.)	POSTOP. B.P. ARMS	DIRECT B.P. UNDER SEDATION ABOVE	DIRECT B.P. UNDER SEDATION BELOW	COMMENT
M. D.*	12	F	165/90	0	Long tapering coarctation	4	140-150/80	122/74	112/72	Small anastomosis (<1 cm.); result gratifying; diminished femoral pulses
H. H.	12	M	180/100	130/?	Total coarctation, vicinity of coarctation hypoplastic	3½	160/80-180/86	152/92	142/90	1.5 cm. area of 50 per cent narrowing; strong femoral pulses, persistent tachycardia; B.P. falls with rate
W. H.	27	M	220/110	0	Hypoplasia of proximal aorta	4	150-180/90	175/100	176/110	No narrowing; strong femoral pulses; small aortic arch; very rapid flow
E. A.	29	M	210/106	130/120	Typical; no hypoplasia	2	110/60-180/100	118/68	104/72	Mild A.I.; minimal narrowing at anastomosis; excellent femoral pulses
R. T.	30	M	240/90	130/110	Typical; lower thoracic aorta small	3	186/72-190/80	146/85 L. vent.	144/82 180/15	A.S., A.I.; anastomosis 60 per cent width of arch; full femoral pulses

*Patient M. D., who has already been discussed, is the individual with the relatively small anastomosis.

those above. The proximal aorta was catheterized via the right brachial artery and contrast studies as well as pressure measurements were made. Pressure in the proximal aorta was 122/74 mm. Hg, while that in the femoral artery was 112/72 mm. Hg. The motion pictures show definite evidence of residual narrowing but, in comparison to the small caliber of the transverse aortic arch, this narrowing seems minor and the contrast medium passes this area with high velocity and no suggestion of delay (Fig. 13). It had been contemplated that this girl would require reoperation with grafting in her adult life. In view of these studies, it seems doubtful to us that the improvement to be obtained would be worth the risk entailed. At the present time (Fig. 12), this patient, in spite of her small anastomosis, is not significantly hypertensive. These findings suggested to us that residual narrowing at the site of anastomosis was probably not a major factor in the pathogenesis of persistent hypertension.

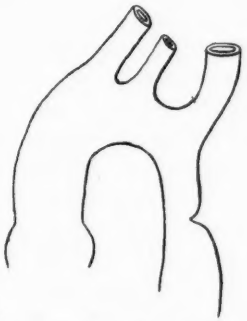


Fig. 14.—Tracing of an enlargement from a 35 mm. motion-picture frame on Patient E. A., aged 29 years, 2 years following surgery. This patient has excellent femoral pulses and a pressure gradient under direct measurement was 14 mm. of mercury systolic. This patient also has mild aortic insufficiency.

As mentioned, 4 other patients were studied in this regard. Their data are summarized in Table II. In spite of the residual hypertension, all of these patients had excellent femoral pulsations on examination and, under essentially basal conditions with sedation, directly measured gradients across the area of coarctation are small, the maximum being 14 mm. of mercury systolic pressure. In 2 patients there was significant detectable residual narrowing. One of these at the time of operation had very definite hypoplasia in the immediate vicinity of the coarctation which undoubtedly accounts for this finding. The other one does have some residual deformity at the anastomotic area (Fig. 14).

In 2 patients, contrast medium was seen to regurgitate across the aortic valve in diastole, confirming the presence of mild aortic insufficiency which undoubtedly contributes to their systolic hypertension.

In 3 of the 4 patients, at the time of surgery the small caliber of various portions of the aorta was noted. This finding was also noted by individuals doing the recheck contrast-medium studies.

The team that studied these patients was also most impressed by the high velocity of flow in the aorta. This is readily demonstrable with the motion pictures obtained using the photofluorographic unit and image intensifier. The finding of relatively small aortas and high velocity flow suggests that a large component of the hypertension in these patients is related to a small inelastic central arterial reservoir, so that the hypertension is not unlike that seen in the arteriosclerotic person, that is, chiefly a systolic hypertension with a relatively normal diastolic pressure. It is likely that there is deficient or abnormal elastic tissue in many of these aortas. However, we do not have sufficient histopathologic proof of this at the present time to warrant further discussion. If patients with coaretation are considered frequently to have a small central arterial volume and decreased vascular elasticity, their blood pressure might be expected to vary roughly in proportion to variations in heart rate. The thought is ingrained in these patients that their well-being is closely allied to low blood pressure; hence, the mere application of the blood pressure cuff provokes an emotional stimulus that may result in tachycardia. Thus, it is not surprising to get widely varying and frequently hypertensive readings in postcoarctation patients. Of the 4 hypertensive patients discussed here, 2 are hyperkinetic and, at almost every examination, have had rather marked tachycardia. In both, blood pressure has responded rather dramatically to Rauwolfia preparations that have also slowed the heart rates.

The 4 patients restudied for hypertension are male; we do not have a clear-cut example of this postoperative problem in female patients. This suggests that the male propensity for arteriosclerosis may be accelerated by coarctation. In this limited series, the age of the patient seems to have little bearing on the persistence of hypertension.

We do not propose here to settle the dispute concerning the pathogenesis of hypertension in coarctation of the aorta. There are proponents of simple mechanical obstruction, and also of renal ischemia.[17] We suggest that postoperative residual hypertension may be largely explained on the basis of the structural characteristics of the blood vessels, and that comparable factors must certainly be applicable preoperatively. In the course of re-evaluating the postoperative conditions of the 5 patients who have been discussed, it was believed to be worth while to study their renal function rather closely. It was suggested that perhaps such a patient might have a second coarctation-like lesion involving the renal circulation to maintain his hypertension. Also, if these patients had typical essential hypertension, it should be manifested in the renal circulation. Mere observation of the patients under hospital conditions was enlightening. It was apparent that this was a systolic and not a diastolic hypertension, as are both essential and renal hypertension. Furthermore, under hospital conditions, all 5 patients became essentially normotensive, which should not happen to a person having essential or renal hypertension without specific treatment. The renal function tests on these patients are summarized in Fig. 15 and Table III. Renal plasma flow and glomerular filtration rate was normal or elevated when compared to average

normal values.[18] Filtration fraction was normal and total renal vascular resistance was normal or slightly decreased. Thus, none of these patients evidenced the renal vasoconstriction characteristic of essential hypertension. Our results are in accord with those which Kirkendall and associates[19] found prior to the surgical repair of aortic coarctation. Such findings suggest that the hypertension of coarctation is not of humoral origin.

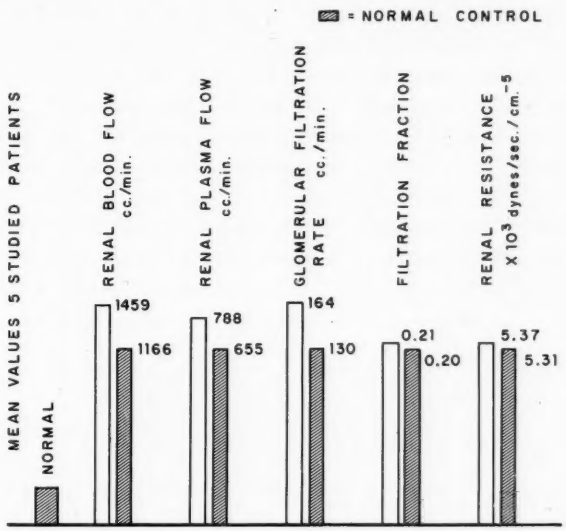


Fig. 15.—Graphic demonstration of mean renal function values in 5 postoperative patients tabulated in Table III. There is no evidence of renal vasoconstriction in these postoperative patients.

TABLE III. TABULAR SUMMARY OF RENAL FUNCTION STUDIES IN 5 POSTOPERATIVE PATIENTS WITH MEAN VALUES AND COMPARABLE NORMAL VALUES[18]

NAME	RBF	RPF	GFR	FF	PM	R
E. A.	1,640	902	208	0.23	100	4.37
M. D.	1,877	1,070	164	0.15	107	4.11
H. H.	1,113	612	133	0.22	100	6.42
W. H.	1,366	683	186	0.27	120	6.39
R. T.	1,295	675	129	0.19	101	5.58
Means	1,459	788	164	0.21	106	5.37
Normal[18]	1,166	655	130	0.20	100	5.31

RBF = renal blood flow in c.c. per min.; RPF = renal plasma flow in c.c. per min.; GFR = glomerular filtration rate in c.c. per min.; FF = filtration fraction; PM = mean arterial pressure in mm. of mercury; and R = renal resistance in 10^3 dynes/sec./cm.$^{-5}$

Note that in every instance the values fall within the normal range and that the mean values exceed normal.

We suggest that coarctation of the aorta as a congenital anomaly consists of not only the narrowed segment, but also of other integral defects of the aortic arch system. These defects are hypoplasia of varied caliber and extent, and deficiencies of the inherent aortic elasticity. Admittedly, this latter defect is largely hypothetical, but more than premature development of arteriosclerotic rigidity is probably present in the young persons studied. The

narrowed segment only can be coped with surgically, and other factors cause residual hypertension. They are probably also major factors in the preoperative hypertension.

SUMMARY AND CONCLUSIONS

The purpose of this paper has been to discuss problems that have continued to perplex the authors in the course of their surgical experience with coarctation of the aorta in 60 patients. There are other aspects of the problems that could have been discussed, but those cited have seemed to be the major ones in that they recur with significant regularity. A discussion of this type does not warrant many firm conclusions and the primary mission when this project was initially undertaken was to clarify, rather than solve, the problems under discussion. However, the clinical research involved in assembling these data has led to the following tentative conclusions.

1. An aggressive surgical approach to the infant patient in congestive heart failure with coarctation of the aorta is indicated. In a majority of these patients, the coarctation is only a part of a complex cardiac anomaly;

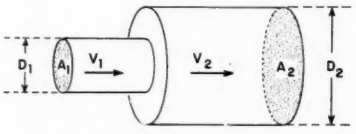


Fig. 16.—See text.

however, the coarctation and the frequently associated patent ductus are the most accessible parts of such problems from the surgical standpoint and operation has resulted in very gratifying results. Residual aortic hypoplasia seems to be well tolerated.

2. No conclusions can be drawn about surgical treatment of coarctations in patients more than 45 years of age. The surgical risk is certainly high, but each case must be weighed on its individual merits.

3. Experience with the postcoarctation abdominal pain syndrome is very limited. It is our impression that this complication is probably associated with extremely complete correction of coarctation and maximal increase in visceral circulation. We hope that additional reports of treatment with antihypertensive drugs will be forthcoming.

4. We believe that a strong case can be made for satisfaction with moderate residual anastomotic narrowing. It seems reasonable to avoid the use of grafts if an anastomotic lumen of 1 cm. in diameter can be accomplished without their use.

5. Postoperatively, these patients tend to have a labile blood pressure and it is thus difficult to ascertain which patients are truly hypertensive; in this series, the figure approximates 10 per cent. Study of such patients suggests

that the hypertension is due to a small inelastic central arterial reservoir rather than residual obstruction and further suggests that, in all probability, the same factors play a role in the hypertension of the untreated patient.

APPENDIX

It is of interest to consider the hydraulic aspect of fluid passing through a constriction. This has been worked out mathematically for the flow of homogeneous fluids through rigid tube systems. The problem becomes much more complicated when one considers a corpuscular fluid, such as blood, flowing through elastic vessels in a pulsatile manner.

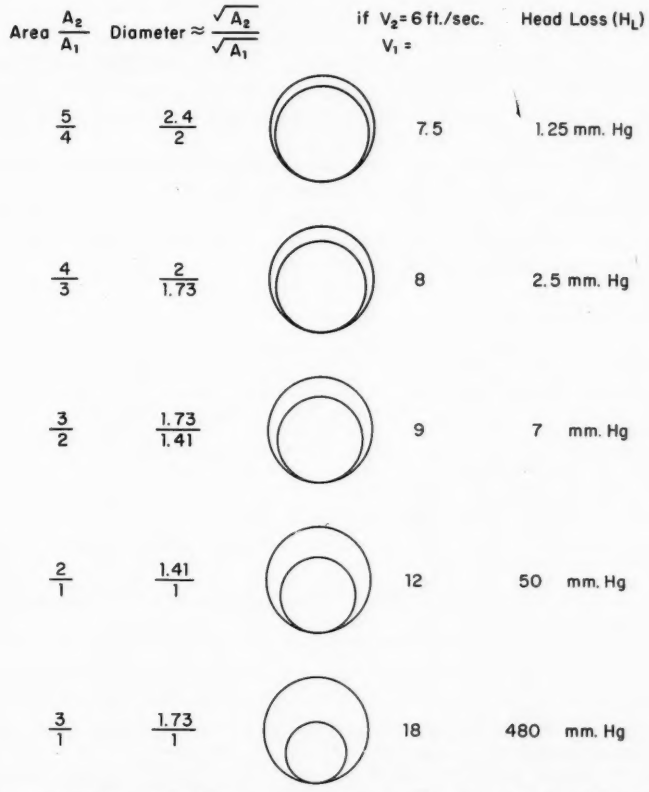


Fig. 17.—"Head loss" associated with constrictions of varying magnitude. The circles correspond to the area and diameter ratios given.

The discussion which follows is thus obviously a gross oversimplification of the problem as it applies to a steady flow of water in a rigid tube system.

There is very little energy loss in a closed system from fluid entering a sudden constriction. The loss is associated with the exit from this constriction where there is a sudden loss of velocity and much energy is lost from turbulence (Fig. 16). This "head loss" may be expressed mathematically by the following equation:

$$H_L = \frac{V_1^2}{2g}\left[\frac{A_2}{A_1} - 1\right]^2 \frac{d}{144}.$$

In this equation H_L = "head loss" in pounds per square inch; V = velocity in ft./sec.; g = the acceleration constant of gravity (32 ft. per sec.^{2}); and d = density of water (62 pounds per cu. ft.).

To assist in solving this equation and further to simplify grossly the problem, several assumptions are made.

V_2 is arbitrarily assumed to be 200 cm. per second which equals approximately 6 ft. per second (a not unreasonable value at the aortic valve).

1 lb. per sq. inch approximates 50 mm. of mercury; therefore a pressure of 150 mm. of mercury approximates 3 lb. per sq. inch.

V_1 is related to V_2 by the following equation:

$$V_1 = V_2 \frac{A_2}{A_1}.$$

Keeping V_2 a constant (6 ft. per second) and canceling out the density of water (62 lb. per cu. ft.) against 2 g (64 ft. per second^2) as equal to essentially unity, the equation is now solved for several common ratios of cross-sectional areas for A_2 and A_1 (Fig. 17). Note that the "head loss" is not great until the area ratios acquire an almost 2 to 1 discrepancy. It is of interest that this rough mathematical analogy is in essential agreement with the findings on a clinical and laboratory basis in vivo.

We are indebted to Mr. Frederick Olmsted, Research Division, The Cleveland Clinic Foundation, and Professor Ray Bolz, Department of Mechanical Engineering, Case Institute of Technology, Cleveland, Ohio, for assistance in this mathematical analysis.

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Discussion

(Papers by Burford [page 47] and Groves [page 60] and their Associates)

DR. JOHN C. JONES, Los Angeles, Calif.—I would like to say that I thoroughly agree with all Dr. Ferguson, Dr. Groves, and their respective coauthors have said.

[Slide] I would like to discuss some of the results on our patients in our experience over the last 12 years. My associates and I have operated on 230 patients up until January 1 of this year.

You will note that there were two deaths in the 14 patients under the age of 1, and 10 of those 14 patients were 10 weeks or younger, and the two deaths were in infants 4 and 5 weeks of age.

This is a miserable group. Nevertheless, I think it is worth while doing everything that can be done. We don't advocate that these patients be catheterized because they are usually in failure and are very sick children.

The vast majority of the patients that we have operated upon were between the ages of 6 and 30 years. Eight of the patients were between the ages of 41 and 49 years and there was no mortality in the older group. Two of them were 49 years of age, and all of them have done well following surgery. There were 2 patients in this group who were severely incapacitated before we operated on them, and they were completely rehabilitated later after their operation.

Fifteen of the last 100 patients, or 15 per cent of the last 100 patients on whom we have operated, have received homograft implants following resection of the coarctation. Before we had grafts available to us we did 11 subclavian-aortic anastomoses with what we thought were very satisfactory results. In these two categories we have had no deaths and no particular morbidity.

Included in the mortality of 3.9 per cent of these 230 patients were 3 patients who developed mycotic aneurysms at the site of the anastomosis. In other words, 33 per cent of the mortality has been due to mycotic aneurysm at the site of anastomosis. Two of these have occurred in the last 6 months and the other one was several years ago.

These 2 that we had recently were boys, ages 16 and 6. They occurred after a long period of no mortality. Neither had pleural or wound infections and the source of their infection is not known. The 16-year-old boy had acne; he returned to the hospital after having been discharged from the hospital on his eighth postoperative day in what we thought was satisfactory condition. When he came back he had fever and x-ray study revealed an aneurysm; he did not respond at all to intensive therapy, and he died of rupture of a mycotic aneurysm. His blood cultures were positive for staphylococcus. The 6-year-old boy operated on 2 months later returned to the hospital after having been discharged on the seventh postoperative day. His mycotic aneurysm did not respond to intensive chemotherapy and it ruptured. Blood cultures were negative. Now in retrospect, this young boy had a history of frequent respiratory infections, having been off and on antibiotics for 6 months prior to the surgery. This experience would indicate that these patients should be free of all infections without antibiotic therapy for at least 6 weeks before any cardiovascular surgery

is undertaken. I believe the increasing incidence of resistant infections and remote resistant infections harbored by these patients is going to lead to more frequent complications of this sort, and this is a complication that is most difficult to deal with.

You will note that we have not carried out the subclavian aortic anastomosis since we have had grafts available, but nevertheless we have felt that these results over a long-term period have been quite satisfactory.

[Slide] Recently we had a patient with a subclavian aortic anastomosis return because she was having dizziness, and it afforded us an opportunity to do a retrograde catheterization via the femoral artery. Unfortunately the dye study is not good, but in the roentgenogram itself it can be seen that at the anastomosis there is quite satisfactory lumen in spite of the fact that this girl had a hypoplastic transverse arch at the time of her surgery. She still has some residual hypertension; but nevertheless the mean pressure readings at the time of her aortograms was 110 mm. Hg, both above and below the anastomosis. It was decided by the cardiologist before we did this that her dizziness was on the basis of hyperventilation.

DR. A. G. BROM, Leiden, The Netherlands.—I am very grateful to you for giving me the opportunity to attend this meeting and to listen to so many interesting papers. I especially want to congratulate Dr. Groves and Dr. Ferguson for their investigation. I should like to make two remarks about the problem of coarctation.

First of all, upon the size of the anastomosis. [Slide] On this slide, you see on the ordinate, the quotient between the systolic pressure in the femoral and brachial artery, taken intra-arterially. So, point 1.00 means that the systolic pressure is the same in both arteries. At point 1.10 the pressure in the femoral artery is higher while at 0.070 it is lower, etc. I want to emphasize that these findings are relative and have nothing to do with the absolute value of the pressure readings.

On the abscissa is shown the quotient between the surface of the anastomosis and the aortic arch as calculated, approximately, during the operation. At point 1.00 the surface is the same. At 1.10 it is more, etc. As should be expected from earlier investigations, there is only a slight difference in systolic pressure between the brachial and femoral artery as long as the anastomosis is more than 50 per cent of the aortic arch. More interesting, however, is the finding that in patients in whom the surface of the anastomosis is larger than the surface of the aortic arch, the systolic pressure in the femoral artery might be lower than in the brachial. After finding this, we went over our pictures made during the operation and then got the impression that the result of an operation for coarctation doesn't always depend upon the absolute size of the anastomosis, but also on the form of the reconstructed descending aorta. If the size is more than 4 cm. in circumference, which means about 1.3 cm. in diameter, then we think this will be sufficient in an adult.

[Slide] But, there should not be any residual waistline in the aorta; the reconstruction of the arch and descending aorta should be so that the medioventral side of the aorta becomes a smooth curve, and the nearly always present bulging of the aorta on the dorsal side should disappear. To accomplish this, we either resect more or make an incision in the bulging part. The use of hypotensive drugs and an approximator helped us to get this shape of the aorta and, at the same time, to obtain an anastomosis with a circumference of 4 cm.

I don't want to say that my fish is bigger than Dr. Jones's fish! But, we operated upon more than 300 patients and by following the above-described technique, we used only thirteen grafts.

Now, I want to say something about the histology of the coarctation. At our University, Dr. Wielenga has made serial sections of the resected specimens and he has found that there is a close relation between the ductus arteriosus and the coarctation, regardless if the ductus is patent or not. There is marked difference between the collagen-rich, elastin-poor, fibrous tissue of the ligament and the elastic tissue of the aorta. The extension of this ductus tissue is pretty much the same in all investigated specimens. It goes from the

point of insertion around the whole of the stenotic aorta. On the ventral side, and at the point of insertion of the ductus into the aorta, it makes up for the whole of the media of the aorta and on the lateral and dorsal walls it is between the intima and the media.

[Slide] Right here is the insertion of the ductus and you see this typical, elastin-poor, fibrous ductus tissue making up the whole of the media of the aorta, and on the opposite side you will find that the same tissue is present between the media and the intima of the aorta. Dr. Wielenga could locate this fibrous ductus tissue in many serial sections and it followed always the same pattern—from the place of insertion, inside the media, at the site of the stenosis, around the aortic wall, regardless if the ductus was patent or not. This ductus tissue has also been found in normal aortas, but there it is limited only to the place of insertion of the ligament.

Thank you once again for the privilege of entering into this discussion. I apologize to the members of the Society for taking more than 3 minutes of their time, but the subject of coaretation of the aorta is not only near to the heart of the patient, but also very close to my own.

DR. GEORGE H. HUMPHREYS, New York, N. Y.—I, too, appreciated very much these two papers, and hesitate, after the large series of Dr. Jones and Dr. Brom, to bring the results of our smaller series to you, but I feel that this is a good chance, as Dr. Groves brought out, to compare notes.

Since 1947, when we started doing coarctations at the Presbyterian Hospital in New York, we have had 86 cases. Our results have been remarkably similar to those reported, with one exception. We have not found many in the infantile group, in spite of the fact that, since publication of the first report from St. Louis, our pediatricians have been very much aware of this situation and have been looking for such patients.

We have, as a matter of fact, operated upon only 3 infants less than 3 years old. There were no deaths in this group, but 1 of these patients did have a recurrence of his coarctation 5 years later.

I would like to ask whether either Dr. Ferguson or Dr. Groves has seen any recurrent coarctation in any of their cases done in infancy.

We were aware, of course, of the growth factor. These operations were done with interrupted sutures of fine catgut in the hope that growth would not be interfered with. The patient whose coarctation recurred was operated on again successfully and is doing well. Incidentally, none of these patients had any other lesions.

In regard to the older age group, like the series from Cleveland we have had 3, 2 aged 47 and one aged 45. One of these was corrected with a bypass graft and the other 2 were done by direct anastomoses. One of the patients died of coronary occlusion 6 weeks after successful recovery from operation.

This brings up the question as to whether reduction of hypertension in the proximal aortic arch in a patient with arterial disease is really a good idea or not.

Like Dr. Jones, in the early period we resorted to subclavian turndown. [Slide] We have here a postoperative angiocardiogram of a patient who had such an operation done approximately 2 years before this picture was made. This shows that the subclavian remained small; the anastomosis is approximately only 1 cm. in diameter. This patient was an adolescent girl and later became a professional tamer of wild horses. She had no difficulty whatever physiologically.

We have done 11 subclavian turndowns. Most of the patients do have some residual hypertension. We have done, since this operation became obsolete, 12 grafts, and many of these patients also have some hypertension remaining.

[Slide] This is a study done on a patient with a graft who did have some residual hypertension; as you see, 168/64 mm. Hg after operation, with a differential of 125/55 in the femoral artery. This patient had a homograft which became calcified, and I believe this lends support to the concept that it is not a reduction in lumen size but perhaps the rigidity of the wall in the intervening vessel which may play some part in the residual hypertension, since so far as we know the lumen was not compromised in this patient at all.

It also shows that, whereas, before operation there was a lag period in time between the brachial and the femoral simultaneous pressure readings, after operation this lag was eliminated and physiologically this patient is in good shape.

[Slide] The final slide will illustrate the slow return of patients to full recovery. This is as measured by ballistocardiogram, which we feel gives a good indication of the mechanics of recovery from coarctation. The K wave here is not present in the preoperative coarctation. It appears immediately postoperatively in a small form, but gradually gets bigger, and not until 1 year later is it of approximately equal size to the I wave, which is the normal situation, so that, even though the anatomy is restored essentially to normal immediately, there is a rather long, slow recovery period.

There are many other areas in which I would like to compare notes, but time does not permit. Our over-all mortality was approximately 5 per cent, 3 patients dying in the early period of problems which I think would no longer bother us; one immediate death when the clamps were removed, one of hemorrhage due to an inadequately tied intercostal, and one of overtransfusion.

Two late deaths have occurred, one of coronary occlusion as I have already mentioned, and one of mycotic aneurysm similar to those Dr. Jones described.

DR. NATHAN K. JENSEN, Minneapolis, Minn.—The discussions by Drs. Jones and Brom and Dr. Humphreys have been timely.

I wish to record a brief case report dealing with repair of coarctation in the over-45 age group. I think Dr. Humphreys' remarks were very good in this respect. If the individual has advanced generalized arteriosclerotic disease, the reduction of the hypertension may present a hazard. However, if the individual does not have evidence of generalized arteriolar sclerosis, then we feel that it is safe to reduce the hypertension by excision of the coarctation.

This was well illustrated by one of our patients who confessed to 58 years of age. Her husband thought she was 50. She had successfully perpetrated this little deception for 30 years. This woman was known to have severe hypertension for at least 10 years prior to her referral for surgery. Her blood pressure during that time ranged from 230/100 to 250/120 mm. Hg. Physical examination revealed no evidence of generalized arteriolar sclerosis. The eye grounds were normal, and the electrocardiographic studies showed only left ventricular preponderance. There was no evidence of cerebral damage.

Surgery revealed an uncalcified tough vessel free of atheroma and easy to work with. Correction of the coarctation was readily accomplished. The patient's blood pressure was normal immediately after surgery and is now, 3 years later. She remains well and has had no sequelae.

DR. EGBERT H. FELL, Chicago, Ill.—The Society has been greatly privileged to hear these two papers, and I was particularly pleased to hear Dr. Ferguson and associates make a differentiation between coarctations of the preductal and the postductal types occurring in infancy. We agree with their report.

The preductal group, we believe, should be cared for in infancy. Our experience is limited and sad. No patient in the postductal group, diagnosed during infancy, to date has required surgery. We have had one death; an infant died of heart failure within 24 hours of admission to the hospital.

Dr. Gasul and his associates have diagnosed and watched a good number of these patients and have kept them well above the age of 3 to 6 years without a mortality.

[Slide] A few slides will, I believe, confirm and contribute to Drs. Groves and Effler's paper. This drawing was made at surgery 12½ years ago. There is a 4 cm. long stenotic area at the isthmus and a stenosis of the subclavian artery at its origin. The patient is now 25 years old and living a normal life. At surgery, mobilization of the aorta was accomplished without injury to the collaterals and the resulting end-to-end anastomosis was about two thirds the size of the proximal lumen of the aorta. The blood pressures in the right arm and legs are equal, that in the left arm, obviously, remains lower. This patient was

operated upon before aortic grafts were used. I believe this long-term result should encourage us to attempt more direct end-to-end anastomoses and less grafting in young individuals.

[Slide] This slide shows a coarctation occurring in a 21-year-old woman with a golf ball-sized aneurysm just distal to the coarctation, and a slightly smaller aneurysm of the second intercostal artery near the apex of the left chest cavity. At surgery, 3 years ago, the aneurysms and coarctation were resected and excellent approximation and end-to-end anastomosis were accomplished without the use of a graft. This lady since has had a child and is living normally.

[Slide] This drawing shows a 10 cm. long coarctation in a 16-year-old boy. We believed at first a graft would be required, but behind this large subclavian artery in the apex of the left chest cavity was this tortuous aorta. The left common carotid was proximal and the tortuous portion ended at the subclavian artery. The aorta was divided at the junction with the subclavian and the tortuous portion brought out from behind the vagus and recurrent nerves. The 10 cm. hiatus was easily bridged. The patient had a satisfactory postoperative course.

[Slide] This slide shows the pathology of one of the 2 patients who required a graft. This long coarctation beginning just above the diaphragm to below the renal arteries was left in place and a homograft was used to bypass it, using and end-to-side anastomosis at both levels. The preoperative blood pressure in the upper extremities was 260/130 and postoperatively it continued high to 200 mm. Hg systolic and about 260 mm. Hg in the lower extremities (a gain of 160 mm.). Much time was spent studying this patient's kidney function and there was some diminished kidney function on the left side. Exploration of the vessels of the kidneys showed minute blood vessels going to the left kidney and normal vessels on the right. The left kidney was removed, following which the blood pressure came down to 170/95 mm. Hg. Just before leaving Chicago, I learned that this boy was recovering from a cerebral accident.

[Slide] This aortogram shows another very difficult and rare coarctation. This, we believe, is an acquired coarctation. Starting just distal to the left subclavian artery, the aorta was hard, nodular, and stenotic. The aortogram shows a narrowed, irregular lumen to the iliac arteries. We explored this woman 5 years ago, hoping that we might be of help to her. We could do nothing. Two months ago she was seen and was free of most of her coronary pain, but continued with severe hypertension, and no blood pressures were obtainable in the legs or the right arm. She may have "pulseless disease." Time does not permit further discussion of this unusual problem.

DR. GEORGE H. A. CLOWES, JR., Cleveland, Ohio.—I hesitate to bring an even smaller series before the Society, except that we have had an experience that might be of interest to others.

About 8 years ago we were asked to deal with a boy who was then 8 years old. He had a long coarctation with a hypoplastic left arch and a hypoplastic left subclavian artery. We had no graft available to us, and felt that we would like to do something.

A clamp was placed on the left common carotid artery. We waited 20 minutes and nothing happened. The anesthetist assured me he could feel a pulse in the left carotid. Obviously the collateral circulation was quite good. We divided the carotid and turned it down to make an anastomosis to the distal aorta. The boy's blood pressure slowly came back to normal and has continued to be so for the 8 years. He plays football and is in every respect a perfectly normal fellow.

I think that this is of importance primarily, that a piece of normally elastic vessel was put in there which perhaps is rather better than a stiff piece of homograft that might be by this time rather smaller than the normal vessel which has grown gradually in size.

We did study this chap by arterial punctures above and below the anastomosis. Both the pulse tracings are normal and pressures are normal.

DR. FERGUSON (Closing).—One specific question was asked by Dr. Humphreys. We have had no recurrences of coarctation, but the series is yet young. The average follow-up time now is only 2 years in the 8 children.

We have a group of children, of course, in whom the diagnosis of coarctation was established during infancy, but who were not in heart failure. These children were followed until a more satisfactory time; that is, 8 or 9 years of age, and repair done then. It is interesting that no child in this group had an intracardiac shunt or an associated patent ductus.

The most illuminating thing to us about this series is the fact that if an infant comes into the hospital in heart failure and an intracardiac shunt is diagnosed, then it certainly behooves the pediatrician and the surgeon to look for an associated coarctation, because these children can be saved by prompt surgical correction of the coarctation.

DR. GROVES (Closing).—Dr. Jones, we have operated upon only 3 patients over 45. One of these died of mycotic aneurysm about a month later; one is living and well, and the other, with regard to what Dr. Humphreys said, got a beautiful reduction in blood pressure, but when he died a week later his brain was mush. He, too, could not tolerate the successful lowering of the blood pressure with his arteriosclerosis.

The longest follow-up in our infantile group is 4 years. We have not recognized the clinical recurrence of coarctation. We have several other postoperative angiocardiographic studies similar to the ones I have shown which show a residual hypoplasia comparable to what was there at the time of surgery, but we are not aware that true coarctation has recurred.

AN ANALYSIS OF DEATHS FOLLOWING CARDIAC SURGERY

James L. Harrison, M.D. (by invitation), Julio C. Davila, M.D., Bart D. Iaia, M.D. (by invitation), and Robert P. Glover, M.D., Philadelphia, Pa.*

CARDIAC SURGERY has been practiced for only a few years and has stimulated remarkable progress in diagnosis, anesthesia, and in surgical thought and technique. Through the years, new surgical methods, arbitrary classifications of cardiac disease, and indications for operative treatment have been proposed. As these and the results of their application have improved, many interpretations and conclusions based on considerable experience, but sometimes on older concepts, have made their way into the literature, nearly always emphasizing the successes. As the era of open heart surgery comes into its own, it seems advisable to review at least the most obvious failures resulting from closed heart surgery in search of explanation and clarification for what often appeared to be perplexing problems, to attempt to sift out misconceptions and errors in judgment, to define limiting physiologic and pathologic factors as they exist today, and to re-evaluate certain areas where knowledge is obviously lacking.

Table I shows the total number of thoracic operations, total hospital deaths, both medical† and surgical, the number of closed heart surgical operations, and the total number of closed heart surgical hospital deaths that have occurred from December, 1952, through December, 1957, on the services of the Glover Clinic group at the Presbyterian Hospital in Philadelphia. This report is particularly

TABLE I. SUMMARY OF CASES IN THIS STUDY

	1952	1953	1954	1955	1956	1957	TOTALS
Total admissions	1	82	142	231	241	534	1,231
Total thoracic operations	1	71	98	165	188	403	926
Total hospital deaths (med. & surg.)	0	15	11	28	17	27	98
Total closed cardiac operations	1	66	76	120	126	155	544
Total closed cardiac surgical deaths	0	12	6	23	12	14	67
Per cent mortality of closed cardiac surgical deaths	0%	18%	8%	19%	10%	9%	12%

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Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

*Research Fellow of the American Heart Association, 1958-1959.

†Patients admitted to the cardiovascular service for study and evaluation, but who were not operated upon.

concerned with 67 closed heart surgical deaths, listed consecutively without omission, which represent the entire mortality experience of this group for this type of surgery in this institution for this 5-year period.

Table II lists the 544 closed heart surgical operations and 67 deaths that occurred over the 5-year period. It will be noted that pulmonary valvotomy, closure of patent ductus arteriosus, and exploratory cardiotomy have been attended with no mortality. On the other hand, mitral commissurotomy has had a 7 per cent mortality; aortic commissurotomy, 21 per cent; mitral purse-string, 35 per cent; and miscellaneous operations, 18 per cent.

TABLE II. CLASSIFICATION OF DEATHS BY OPERATION, 1952-1957

OPERATION	NO. OPERATED CASES	DEATHS	% MORTALITY
Pulmonary valvotomy	17	0	0
P.D.A. ligation or division	19	0	0
Exploratory cardiotomy	32	0	0
Mitral commissurotomy	275	20	7
Aortic commissurotomy	57	12	21
Mitral purse-string	54	19	35
Miscellaneous*	90	16	18
Totals	544	67	12

*Resection coarctation of aorta; pericardial poudrage; pericardiectomy; infundibular resection; tricuspid commissurotomy; closure interatrial septal defect; excision left ventricular aneurysm; and Hufnagel valve insertion.

Table III is a breakdown by disease in the closed heart surgical group. In the aortic stenosis and mitral insufficiency groups, in which aortic commissurotomy or mitral purse-string operations were performed, 3 and 6 cases, respectively, had associated mitral stenosis for which mitral commissurotomy was also performed. Table IV lists the cases according to clinical classification[1, 2] and sex.

TABLE III. CLASSIFICATION BY DISEASE
PRIMARY HEART DISEASE—67 CLOSED CARDIAC SURGICAL DEATHS

	NO. CASES	SUBTOTAL
Congenital		
Interatrial septal defects (2,* 1†)	5	
Coarctation of aorta	2	
Aortic stenosis	1	
Infundibular stenosis (tetralogy of Fallot)	1	
		9
Acquired		
Rheumatic heart disease		53
Mitral stenosis	20	
Aortic stenosis (3†)	11	
Mitral insufficiency (6†)	19	
Tricuspid, mitral and aortic stenosis	1	
Aortic and mitral insufficiency	1	
Myocarditis	1	
Arteriosclerotic heart disease		4
Postmyocardial infarction (angina)	3	
Left ventricular aneurysm	1	
Unknown		
Constrictive pericarditis	1	1
Grand total		67

*Anomalous pulmonary veins.
†Associated mitral stenosis.

TABLE IV. DISTRIBUTION OF CASES BY CLINICAL CLASSIFICATION AND SEX

NEW YORK HEART CLASS	MALES	FEMALES	TOTALS	AM. ASSOC. ANES. CLASS	MALES	FEMALES	TOTALS
IIC	1		1				
IIIC	2	2	4				
IIID	8	13	21	III	8	16	24
IVD	11	15	26	IV	20	23	43
IVE	6	9	15				
Totals	28	39	67		28	39	67

In Table V the cases are grouped according to type of operation and time of death in relation to surgery. Operating room deaths are defined as those which occur any time during the course of anesthesia or surgery in the operating room. Immediate postoperative deaths are those occurring in the first 48 hours following surgery. Delayed postoperative deaths are those occurring after 48 hours but during the period of hospitalization.

TABLE V. GROUPING ACCORDING TO TYPE OF OPERATION AND TIME OF DEATH

OPERATION	TIME OF DEATH			TOTALS
	O.R.	IMMEDIATE	DELAYED	
Mitral commissurotomy	2	4	14	20
Aortic commissurotomy	6 (2*)	4	2 (1*)	12
Mitral purse-string	6	8 (3*)	5 (3*)	19
Mitral and tricuspid commissurotomy		1		1
Hufnagel valve procedure	1			1
Pericardiectomy	1			1
Pericardial poudrage		2	2	4
Closure interatrial septal defect	1	1	3 (1*)	5
Infundibular resection		1		1
Resection coaretation of aorta	1		1	2
Resection left ventricular aneurysm		1		1
Totals	18	22	27	67

*Coexisting mitral stenosis for which commissurotomy was performed.

Table VI is a compilation of clinical and other features for the mitral commissurotomy, aortic commissurotomy, and mitral purse-string groups which comprise 51 of the 67 deaths under discussion. The other 16 cases are considered later in the text.

Mitral commissurotomy refers to the standard operation as described previously,[3, 4, 5] although 9 of the patients had anterolateral commissurotomy only.

The twelve aortic commissurotomies were performed by the transventricular route as described elsewhere.[6] Of the 12 deaths under discussion, 6 of the patients were operated upon with the now obsolete three-bladed dilator[6] and 6 with the two-bladed Brock dilator.[7] The current technique[8] was used in only 2 of the latter. Circumferential suture of the mitral valve was carried out as described in previous publications.[9, 10, 11]

The obviously high mortality rates in the aortic commissurotomy and mitral purse-string groups are, in part, related to the development of these procedures. Better selection, refinement of method, and progress in management and anesthesia[12, 13] have currently reduced these to acceptable rates.[8, 10, 11, 14] This study further represents the group's experience largely in acquired valvular cardiac

disease. The bulk of the congenital heart surgery is performed at St. Christopher's Hospital for Children in Philadelphia and does not form a part of this survey.

DISCUSSION OF MORTALITY IN SURGERY FOR MITRAL AND AORTIC STENOSIS AND MITRAL INSUFFICIENCY

Table VI summarizes most of the information pertaining to the mitral commissurotomy, aortic commissurotomy, and mitral purse-string groups.

Patients with mitral stenosis tolerate surgery much better than those with aortic stenosis or mitral insufficiency, and the majority of those who die do so in the postoperative period of advanced disease or of induced mitral regurgitation. On the other hand, patients with aortic stenosis and mitral insufficiency are faced with a high immediate risk, related particularly to more profound myocardial and systemic changes. It is apparent from the distribution by classification,* in all categories, that nearly all the patients were in far-advanced stages of disease. This is particularly true in the aortic stenosis and mitral insufficiency groups.

The ratio of males to females who die in the mitral commissurotomy group is higher than for the over-all series.[15] In contrast, the number of males undergoing aortic commissurotomy is about three times as high, and there seems to be no relation of sex to surgical risk in this group.[8, 15] In the mitral insufficiency group, the ratio of males to females undergoing surgery is about the same as for those who die.[14, 15]

The age of the patient undergoing mitral commissurotomy may be related to the surgical risk in that the median age among the fatal cases is 4 years higher than in the over-all group of 275 cases.

The median age of patients with aortic stenosis who died was also 4 years greater than that of the over-all operative material (57 cases). The median age of the group with fatal mitral insufficiency was only 1 year greater than that of the series of 54 patients operated upon.

Although it is implied, in the figures listed, that most of the patients in all categories who died following surgery had rheumatic fever at or about age 15, it must be borne in mind that this figure depends on the patient's recollection of the onset of disease and is probably 4 to 6 years higher than it should be.[16]

It is interesting to note that cardiac symptoms were present for about one third of the total interval since onset of disease. However, frank congestive cardiac failure did not begin until the last 1½ to 3½ years prior to surgery. It

*The Functional-Therapeutic New York Heart Association Classification[1] is as follows: Class I—no limitation of activity; Class II—slight limitation of activity; Class III—moderate to great limitation of activity; Class IV—unable to carry on any activity without discomfort. Class A—no restriction required; Class B—restriction in unusual activity; Class C—moderate restriction of ordinary activity; Class D—marked restriction of ordinary activity; Class E—should be at bed rest.

American Association Anesthesiologists Classification of physical status[2]: Class I—no organic pathology; Class II—moderate but definite systemic disturbance; Class III—severe systemic disturbance; Class IV—external systemic disorders (eminent threat to life); Classes V and VI—emergency states.

TABLE VI. TABULATION OF DATA ON MITRAL COMMISSUROTOMY, AORTIC COMMISSUROTOMY, AND MITRAL PURSE-STRING GROUPS

	MITRAL COMMISSUROTOMY		AORTIC (3*) COMMISSUROTOMY		MITRAL (5*) PURSE-STRING	
	NO.	PER CENT	NO.	PER CENT	NO.	PER CENT
Total cases operated	275		57		54	
No. of deaths and % mortality	20	7.2	12	21.1	19	35.2
No. of deaths	20	100	12	100	19	100
Operating room deaths	2	10	6	50	7	37
Immediate postoperative deaths	4	20	4	33	7	37
Delayed postoperative deaths	14	70	2	17	5	26
Classification, New York Heart Association						
Class IIIC					1	5
Class IIID	10	50	2	17	5	26
Class IVD	6	30	6	50	7	37
Class IVE	4	20	4	33	6	32
Classification physical status (American Association Anesthesiologists)						
Class III	10	50	1	8	7	37
Class IV	10	50	11	92	12	63
History of rheumatic fever or related disease						
Definite	13	65	6	50	15	78
Vague	3	15	3	25	2	11
None	4	20	3	25	2	11
Sex distribution						
Males	7	35	9	75	2	11
Females	13	65	3	25	17	89
Ages (in years)						
Median	44		44		32	
Range	33-63		30-50		18-52	
Number of years rheumatic fever or cardiac disease present						
Median	10		12		6	
Range	5-42		13-42		5-36	
Number of years taking digitalis						
Median	4		2		6	
Range	0-25		0-13		1-11	
Number of years since onset of frank congestive cardiac failure						
Median	2½		1½		3½	
Range	0-10		0-13		1-18	
Congestive cardiac failure on admission						
Frank	9	45	3	25	9	47
Borderline	2	10	4	33	2	11
Not present or fully compensated	9	45	5	42	5	26
Possibility of preoperative rheumatic fever activity	4	20	2	17	6	32
Possibility of or suggested digitalis intoxication	8	40	5	42	11	58

(Cont'd on next 2 pages.)

TABLE VI—CONT'D

	MITRAL COMMISSUROTOMY		AORTIC (3*) COMMISSUROTOMY		MITRAL (5*) PURSE-STRING	
	NO.	PER CENT	NO.	PER CENT	NO.	PER CENT
Electrocardiogram (preoperative)						
SR	2	10	9	75	6	32
AF	17	85	3	25	13	68
Bigeminy	1	5	(2)	(runs)		
RVH	13	65			5	26
LVH	1	5	9	75	4	21
L and RVH	4	20	3	25	4	21
Strain or overload	10	50	6	50	4	21
Partial AV block			1	8	2	11
L.B.B.B.			1	8		
Healed infarct			1	8		
Left ventricular size by x-ray (scale 0 to 4 plus)						
Median	½		3		2	
Range	0-2		2-4		1-4	
Irritable myocardium at surgery	6	30	8	67	14	74
Blood replacement at operation (c.c.)						
Median	650		1,500		1,500	
Range	500-1,500		500-2,500		500-4,000	
Thrombus in left atrium or appendage	11	55	1	8	2	11
Arterial emboli produced						
Definite	6	30	1	8		
Probable	1	5	1	8		
Postoperative shock in those surviving operation						
Immediate postoperative deaths	4 of 4		4 of 4		7 of 7	
Delayed postoperative deaths	2 of 14		2 of 2		2 of 5	
Postoperative frank CHF in those surviving surgery						
Immediate postoperative deaths	1 of 4		1 of 4		2 of 7	
Delayed postoperative deaths	13 of 14		2 of 2		5 of 5	
Mitral regurgitation						
Premitral commissurotomy	11 of 20		2 of 3			
Postmitral commissurotomy	18 of 20		3 of 3			
Increase in mitral regurgitation	17 of 20		3 of 3			
Degree mechanical correction of mitral regurgitation by mitral purse-string						
75% or more					13	68
50 to 75%					3	16
Less than 50%					3	16
Description of mitral valve (operation and autopsy)						
4-plus calcified, rigid	8					
2-plus calcified, 1-plus pliability	2					
Shoeleather with some calcification, rigid	5				2	
Shoeleather with 1-2 plus pliability	3		3 of 3		4	
Thin shoeleather 2-3 plus pliability	1				5	

TABLE VI—CONT'D

	MITRAL COMMISSUROTOMY		AORTIC (3*) COMMISSUROTOMY		MITRAL (5*) PURSE-STRING	
	NO.	PER CENT	NO.	PER CENT	NO.	PER CENT
Kid glove, 4-plus pliability	1				7	
Normal valve consistency					1	
Marked loss of valvular substance					4	
Slight to moderate loss valvular substance					10	
Degree of mitral stenosis						
0.1 to 0.2 cm.²	4	20				
0.2 to 0.8 cm.²	8	40				
0.8 to 1.5 cm.²	4	20				
Over 1.5 cm.²	4	20				
Mitral annulus (diameter in cm.)						
Median					(10 of 19) 6	
Range					5-7	
Mitral orifice (diameter in cm.)						
Median					(14 of 19) 4	
Range					3-6	
Aortic valve						
4-plus calcification			10	83		
3-plus calcification			2	17		
Calcification into myocardium			9	75		
Heart, pathologic findings (number)	(12)		(7)		(15)	
Muscle hypertrophy	8		6		13	
Perivascular fibrosis	8		7		10	
Recent myocardial infarcts	2		1		0	
Healed myocardial infarcts	0		1		0	
Rheumatic activity	1		1		0	
Fatty change	0		1		3	
Endocardial thrombus	3		0		1	
Significant coronary atheromatosis	5		9		5	
Lung, pathologic findings (number)	(15)		(9)		(16)	
Chronic passive congestion	14		9		16	
Pulmonary fibrosis	5		0		1	
Bronchopneumonia	4		1		2	
Pulmonary edema	4		4		9	
Pulmonary infarction	2		0		2	
Liver, pathologic findings (number)	(13)		(8)		(14)	
Chronic passive congestion	9		5		11	
Cardiac cirrhosis	3				2	
Central necrosis	1				3	
Fatty change	1		4		1	

*Associated mitral stenosis requiring mitral commissurotomy.

is notable that the interval of time that patients had congestive cardiac failure with aortic stenosis is considerably shorter than with either mitral stenosis or mitral insufficiency, but it is also evident that patients with mitral insufficiency had a much shorter interval during which symptoms were present. These considerations could support the view that the patient with mitral stenosis has a long course of illness with a relatively slow progression of symptoms and

deterioration. Likewise, the patient with aortic stenosis has a long course of illness with relatively slow development of symptoms but with rapid deterioration after onset of congestive heart failure. The patient with mitral insufficiency has a shorter interval of rheumatic disease, with symptoms developing late in this course, followed quickly by congestive cardiac failure of a less precipitous nature than in cases of aortic stenosis.

The electrocardiographic findings in the three groups in Table VI indicate little that would not be expected in the over-all series; however, atrial fibrillation in the fatalities was more common in mitral stenosis and occurred in aortic stenosis only when there was associated mitral stenosis. Ventricular hypertrophy was overwhelmingly right in mitral stenosis, left in aortic stenosis, but about equally distributed between right, left, and combined in the group with mitral insufficiency. The presence of conduction defects in aortic stenosis and mitral insufficiency seems definitely associated with a higher operative risk.[15, 17] The presence of myocardial irritability during surgery was determined from the operative and anesthesia protocols. It was observed in over half the fatalities, occurring in two thirds and three fourths, respectively, of the aortic stenosis and mitral insufficiency groups.

The median value for blood replacement is definitely higher in the aortic commissurotomy and mitral purse-string groups than in the others.

Thrombus was present in the left atrium or appendage twice as frequently in the fatal cases as in the over-all series for mitral commissurotomy.[15, 18] The incidence of atrial thrombosis is low for the other two categories, as expected. Arterial emboli, producing cerebral infarction, were noted unequivocally in 6 instances and probably in 1 other case.* This is about seven times more frequent than is found in the over-all mitral commissurotomy series.[15, 18] There were noncerebral peripheral emboli in 3 of the cases mentioned above.

Preoperative low-grade rheumatic activity is at times very difficult to diagnose with any degree of certainty, but its presence could not be ruled out in about one third of the fatalities under discussion.

It is well recognized that digitalis leaf or its purified derivatives have toxic properties that are frequently associated with its clinical use.[19] The diagnosis of and the degree of severity of digitalis intoxication cannot often be accurately determined and its presence is therefore listed as a possibility. There was no relation between the length of time of digitalis therapy and the suggestion of toxicity during the hospital course. Of the 24 instances of possible toxicity in the three groups under discussion, atrial fibrillation was present in 15, sinus rhythm in 8, and persistent bigeminy in 1 case.

It will be noted in Table VI that about two thirds of the cases in all three groups were in frank or borderline congestive failure on admission to the hospital. The majority of the remaining patients with mitral and aortic stenosis, and all the patients with mitral insufficiency had been in frank congestive cardiac failure at some prior time. There was evidently a wide variation in the effectiveness of previous medical management.

*One of these was from an autopsy-proved calcific embolus causing an infarct in the right internal capsule.

In this group of cases, there is a considerable range in the completeness of hemodynamic data. Most of the patients with mitral and aortic stenosis, operated upon during the period of this study, were not subjected to cardiac catheterization unless there was some special diagnostic problem. Since the development of safe and reliable left heart catheterization techniques, it is our policy to study all cases of aortic stenosis by this means and to attempt more frequently to estimate degrees of regurgitation coexisting with mitral stenosis.[20, 21] In the cases of mitral insufficiency, at least right heart catheterization was done in all but 1 patient undergoing surgery, and left heart catheterization in about two thirds of them.[11, 22]

TABLE VII. HEMODYNAMICS—MITRAL COMMISSUROTOMY
(PREOPERATIVE CATHETERIZATION DATA AVAILABLE IN 4 OF 20 CASES)

CASE	PA	LA OR PAW	ART.	CI	PAR	REMARKS
a.	60/15(30)		96/22	1.6		
b.	48/28(38)		92/60			
c.	36/20(26)	25/15(20)	160/80	2.3	3.9	
d.	80/40(57)	45/30(35)	180/116	2/4	6/9	MS-MI

Operative Pressures

CASE	LA	LV	AO.	CUFF PRESSURE	REMARKS
b. Preop.	34/12(23)	136/5	105/22	120/80	Severe aggravation
Postop.	60/0 (30)	123/15	95/40	105/80	of MI produced
d. Preop.	38/25	108/12	108/78		MI preoperatively;
Postop.	46/23	155/20			aggravation by surgery

Figures in parentheses refer to mean pressure—all pressures in mm. Hg.
PA—Pulmonary artery; LA—left atrium; PAW—pulmonary artery wedge; Art—systemic artery; CI—cardiac index; PAR—pulmonary arteriolar resistance; Ao—aorta; LV—left ventricle.

In 4 cases of the 20 deaths due to mitral commissurotomy there were preoperative hemodynamic studies which are shown on Table VII. In 2 of these, left heart pressures were measured at surgery. In these, it is worth noting that significant increases in left ventricular diastolic pressure, concomitant with elevation of left atrial pressure, decreased aortic pressure and decreased atrioventricular gradient, all of which suggest severe, acutely produced mitral insufficiency.

Hemodynamic data are available in 6 of the deaths due to aortic stenosis. In 3 of these, operative measurements were made (see Tables VIII—A and B). It is important to note that there was an elevated left ventricular end-diastolic pressure in all of these in the absence of demonstrable aortic or mitral insufficiency. Operative measurements indicate marked reduction in the systolic ventriculo-aortic peak gradient with decrease in the left ventricular end-diastolic and systolic pressures in 2 and decrease in the systolic but marked increase in the end-diastolic in 2. This indicates the production of significant aortic insufficiency in the former and mitral insufficiency in the others.

The hemodynamic data collected in the study of mitral insufficiency patients have been reviewed elsewhere.[10, 11] The information pertinent to this study is summarized in Table IX. Fifteen of the 18 patients had pulmonary artery

TABLE VIII A. HEMODYNAMICS—AORTIC STENOSIS
(PREOPERATIVE CATHETERIZATION DATA IN 3 OF 9 CASES)

CASE	PA	LA OR PAW	LV	AO.	CI	REMARKS
a.	25/?	26/0(14)				
b.	19/2	15/0(10)				
c.	35/8	25/15(20)	200/20	100/70	2.6	

Operative Measurements

CASE	LA	LV	AO.	LV-AO. GRADIENT	REMARKS
b. Preop.	35/6.5(23)	220/13	125/50	95	
Postop.	11/3(6)	150/5	120/60	30	
c. Preop.	62/30	250/44	140/75	110	
Postop.	?	115/52	115/?	0	AI produced

All figures in parentheses refer to mean pressure—all pressures given in mm. Hg.

TABLE VIII B. HEMODYNAMICS—AORTIC STENOSIS-MITRAL STENOSIS
(PREOPERATIVE CATHETERIZATION DATA IN 2 OF 3 CASES)

CASE	PA	LA OR PAW	AO.	CI	PAR	REMARKS
a.	68/16(28)	20/15(18)	65/56	1.4	9	
b. 1953	45/22(30)	33/20(24)	125/64	2.7	1.5	
1954	32/15(22)	(15)		3.1		

Operative Measurements (1 case)

CASE	LA	LV	AO.	A-V GRADIENT	LV-AO.	REMARKS
b. After MC	44/25/(37)	160/14	125/70	23	35	
Not after AC	60/25/(47)	120/21	84/60	26	36	Severe MI

All figures in parentheses refer to mean pressure—all pressures given in mm. Hg.

PA—Pulmonary artery; LA—left atrium; PAW—pulmonary artery wedge; Art—systemic artery; CI—cardiac index; PAR—pulmonary arteriolar resistance; Ao—aorta; LV—left ventricle; LV-Ao—left ventricular aortic gradient; A-V gradient—atrial-ventricular gradient.

TABLE IX. HEMODYNAMICS—MITRAL INSUFFICIENCY DEATHS
(PREOPERATIVE CATHETERIZATION IN ALL 18 CASES)

	AVERAGE	RANGE	DISTRIBUTION		
Pulmonary Artery (mm. Hg) (18 cases)					
Mean pressure	38	22-25	Over 35 (11 cases)	25-35 (4 cases)	Less than 25 (3 cases)
Peak systolic pressure		23-92	Over 50 (11 cases)	40-50 (4 cases)	Less than 40 (3 cases)
Left Atrial or Pulmonary Arterial Wedge (12 cases)	37 / 18 (29)	22-72 / 7-24			
Cardiac Index (12 cases) (L./min./M.²)	2.2	1.1 to 2.8	2 or over (9 cases)		Less than 2 (3 cases)
Operative Pressures					
Left atrium (16 cases)	50 / 19	25-100 / 5-60	Over 25 (all cases)	V wave Over 35 (12 cases)	Over 60 (5 cases)
Left ventricular end-diastolic	15	3-45	Over 20 (2 cases)	10-20 (10 cases)	Less than 10 (3 cases)

systolic pressures over 40 mm. Hg, 11 of which were over 50 mm. Hg. In all but 3 cases there was mean left atrial or pulmonary artery wedge pressures higher than 20 mm. Hg, and there was increased pulmonary arteriolar resistance in two thirds of 9 cases in which this measurement is available.

The description of the mitral valves was derived from the operative and postmortem protocols. The degree of mitral stenosis, the diameter of the annulus, and the size of the orifice were estimated digitally at surgery. Aortic valvular calcification was appraised by external palpation as well as by the degree of resistance to the dilating instrument. In all cases in which autopsy examinations were available, these estimates of calcification have been compared with the postmortem findings and have been found to be generally in agreement.

In every case of mitral commissurotomy, the stenosis was relieved to some degree. However, an adequate commissurotomy as previously defined[4] was performed in only 7 of the 20 fatal cases, and in none of the 3 fatalities of combined mitral and aortic stenosis was it possible to open the mitral valve adequately. Advanced calcification and scarring was the limiting factor in all of these. Among the 7 cases in which satisfactory commissurotomy was possible, 3 patients died in the first 48 hours with cerebral infarction. Of the remaining 4, 1 had myocardial infarction, 1 had pulmonary infarction, 1 had lesser cerebral embolization with a protracted course of bronchopneumonia, and 1 had progressive congestive cardiac failure.

Preoperatively some mitral regurgitation was present in over half of the patients with mitral stenosis and in 2 of the 3 combined aortic and mitral stenosis fatalities. After mitral commissurotomy, mitral regurgitation was present in 18 and 3 of these two groups, respectively. There was definite increase in regurgitation in 17 of the former and 3 of the latter group.

Aortic commissurotomy was carried out, after ventricular fibrillation or cardiac arrest had occurred in 4 instances. A mechanically satisfactory aortic commissurotomy was performed in 10 patients. There was postcommissurotomy aortic insufficiency in 1 according to pressure tracings taken at surgery, and at autopsy in 1 a tear in a cusp with production of a false passage, 1 cm. in diameter was noted.

In the group with mitral insufficiency, the degree of mechanical correction was grossly estimated by digital appreciation of anatomic change in the function of the valve leaflets, change in size, position, and character of the regurgitant jet, and by operative hemodynamic measurements.[10, 11] Mitral purse-string was carried out after the occurrence of ventricular fibrillation or cardiac arrest in 3 cases. In the majority of the fatalities, the mechanical improvement was marked, the limiting factor in a few being the state of the valve, particularly the marked loss of valve substance, downward contraction of valve leaflets, and lack of pliability of the valves themselves. It is apparent that correction or improvement of the mechanical defect is not sufficient, per se, to save many patients in the advanced stage of this disease.[14, 22] Misplaced circumferential sutures, one through the left atrium with subsequent infection,[23] one obstructing the posterior descending branch of the right coronary artery, and one through the base of the aorta represented avoidable technical errors.

The data concerning postoperative shock and congestive cardiac failure in patients surviving surgery refer to the presence of these conditions as part of the postoperative clinical picture for which active supportive treatment was necessary. It is interesting and striking that of the fatalities occurring in the immediate postoperative period all patients showed shock and about one fourth showed congestive cardiac failure. In the delayed postoperative period, shock occurred in about one third of the cases and congestive cardiac failure in 20 of 21.

The actual heart weights in the mitral stenosis and mitral insufficiency groups were about equal, while the group with aortic stenosis had distinctly heavier hearts. It is well known that concentric hypertrophy of the left ventricle is a predominant finding in aortic stenosis while in mitral insufficiency ventricular dilatation is more evident. However, when heart weight is correlated with body surface, this relationship does not hold. It is quite likely that the discrepancy is due to the fact that the vast majority of the patients with mitral stenosis and insufficiency were in advanced states of emaciation which makes the estimates of body surface misleading. Microscopic examination of the hearts showed the findings listed in Table VI. It is interesting to note that in all the cases of aortic stenosis and in one third of the other two categories, there was significant coronary artery disease.

A comparison of heart weights of those patients having cardiac asystole during surgery (Table X), with those who did not, as well as with the groups listed by operation, shows that patients who had asystole did not show significant heart size difference; however, it is noteworthy that in such cases there was a greater elevation of left ventricular end-diastolic pressure.

TABLE X. MEDIAN HEART, LIVER, AND LUNG WEIGHTS

	BODY SURFACE (M.²)	HEART		LUNG		LIVER	
		GM.	GM./M.²	GM.	GM./M.²	GM.	GM./M.²
Mitral commissurotomy (13 cases)	1.50	580	398	950	585	1,500	900
Aortic commissurotomy (9 cases)	1.63	640	370	1,090	675	1,600	1,020
Mitral purse-string (18 cases)	1.54	570	410	730	534	1,600	1,090
Cardiac asystole*	1.54	591	395	952	617	1,730	1,124
No cardiac asystole†	1.56	630	404	874	549	1,480	951

*Left ventricular end-diastolic pressure 22.1 mm. Hg (median).
†Left ventricular end-diastolic pressure 17.8 mm. Hg (median).

MISCELLANEOUS DEATHS

1. *Closure of Interatrial Septal Defects* (5 deaths).—Of the 5 deaths associated with interatrial septal defects (all secundum type), 4 were after Sondergaard closures[24] and one after atrioseptopexy.[25] Two of these patients had anomalous pulmonary veins and 1 had associated mitral stenosis. One death

occurred in the operating room, 1 in the immediate postoperative period, and 3 in the delayed postoperative period. There were 3 females, aged 25, 35, and 41, and 2 males, aged 9 and 41. Four of the patients had symptoms 5 years or longer, 3 of whom were in frank congestive cardiac failure on admission. The pulmonary artery pressures were elevated in all cases, being 38/18, 50/25, 80/36, 86/30, and 100/48 mm. Hg. The first 4 had left-to-right shunts of 4.0, 7.0, 5.0, and 3.6 L. per minute, respectively, but the patient with the pulmonary artery pressure of 100/48 had a bidirectional shunt, the exact values being unavailable for this record. In this case, the pulmonary artery pressure fell to only 92/36 after closure of the defect. The pulmonary arteriolar and total pulmonary vascular resistances in the other 4 cases were 1.7 and 2.9, 2.0 and 2.9, 5.0 and 5.5, and 5.5 and 6.2 resistance units.[26] The electrocardiogram showed right ventricular hypertrophy in 3, right and left ventricular hypertrophy in 2, with right bundle branch block in 1 of these. There was suggestive evidence of digitalis sensitivity in 2 patients, an irritable myocardium at surgery in 3 patients, ventricular tachycardia and fibrillation playing a significant role in death in 3 instances (not merely as a terminal event). Blood replacement in the 4 adult patients ranged from 1,000 to 3,000 c.c. Hemorrhage from an atrial tear followed by ventricular fibrillation was responsible for the 1 operating room death. Persistent ventricular tachycardia and other arrhythmias as well as congestive cardiac failure were responsible for the 1 immediate postoperative death, the arrhythmias being present throughout the entire hospital course and at cardiac catheterization preceding surgery. Of the delayed postoperative deaths, 2 of these patients, aged 37 and 49, had associated congestive cardiac failure and died the tenth and third day, respectively, following surgery. Both had pulmonary artery pressures above 80 mm. Hg systolic. One had elevated pulmonary arteriolar resistance (5.0 units) and mitral stenosis for which a satisfactory digital commissurotomy was performed through the septal defect. The other patient had the bidirectional shunt mentioned above. One postoperative death resulted from congestive cardiac failure following an avoidable technical error which resulted in insufficiency by laceration of the posterior cusp of the aortic valve, as well as creation of an aortico-right atrial fistula. In 4 autopsies, the heart weights ranged from 400 to 640 grams. All showed hypertrophy and fibrosis on microscopic examination. The combined lung weights ranged from 1,420 to 1,720 grams. The lungs showed chronic passive congestion in 4, with edema in 3, and acute congestion in 1. The patient with the bidirectional shunt, who died 10 days postoperatively, also had extensive pulmonary venous thrombosis.

2. *Pericardial Poudrage* (4 deaths).—There were 4 deaths in the patients undergoing pericardial poudrage,[27] 2 in the immediate and 2 in the delayed postoperative period. All were males, the ages being 39, 46, 47, and 57. All were Class IVD according to the classification of the New York Heart Association and Class IV by physical status. One had predominantly rheumatic myocardial insufficiency with minimal valvular disease. The other 3 patients had histories of definite myocardial infarctions, with subsequent angina pectoris.

On electrocardiogram, 1 showed coronary insufficiency, another anterior wall ischemia (the autopsy showed recent and healed myocardial infarcts), and 2 showed left ventricular hypertrophy and nonspecific myocardial damage. Two were in congestive cardiac failure on admission and 2 showed an irritable myocardium at surgery.

Two patients died 7 and 16 hours postoperatively and 2 died 7 and 8 days postoperatively, all with the picture of severe acute myocardial infarction. At autopsy, 2 showed extensive coronary disease with complete occlusion of the left anterior descending artery with healing infarcts in 1 and multiple occlusions in the other.

3. *Resection of Coarctation of the Aorta* (2 deaths).—There were 2 deaths associated with coarctation of the aorta. An 18-year-old boy who died in the operating room, had been symptomatic only 3 months, but had extensive runs of ventricular tachycardia 2 days before operation. Cardiac arrest and ventricular fibrillation occurred 10 minutes after clamping the aorta. Autopsy showed a bicuspid aortic valve with both coronary arteries arising from a common ostium. The other patient, a 36-year-old woman, who had an uneventful operation, was doing well, but unexpectedly died in her sleep on the fifth postoperative day. Perusal of the nurses' notes indicates the strong but unverifiable possibility that digitoxin overdosage through nursing misinterpretation was a contributing factor.

4. *Pericardiectomy* (1 death).—This death occurred in the operating room in a 47-year-old man who died of ventricular fibrillation following hemorrhage from a tear in a tense, greatly distended pulmonary vein. This patient had had a previous attempt at a pericardiectomy and had been in congestive cardiac failure for about 18 years.

5. *Resection of Left Ventricular Aneurysm* (1 death).—This death occurred in a 47-year-old man who had developed a large ventricular aneurysm[29] following myocardial infarction. The resection was carried out without event, although extensive thrombosis was present in the aneurysm. Postoperatively, a left hemiplegia was noted and profuse bleeding occurred through the chest drainage tube. A total of 11,500 c.c. of blood was given and eventually exploration confirmed the impression that the bleeding was not from the ventriculotomy incision but part of a general hemorrhagic diathesis. At autopsy, fibrocaseous pulmonary tuberculosis, adenocarcinoma of the stomach with bone metastases, and mural thrombosis at the ventriculotomy site were present.

6. *Infundibular Resection in a Tetralogy of Fallot* (1 death).—This patient was a very sick 24-year-old woman with long-standing cyanosis and a definite diagnosis of tetralogy of Fallot with infundibular stenosis. The cardiac catheterization data showed a right ventricular pressure of 100/4, infundibular chamber pressure of 40/8, and pulmonary artery pressure of 16/8 mm. Hg. The peripheral oxygen saturation was 49 per cent. The preoperative hematocrit was 81 per cent and the hemoglobin 22.6 grams per 100 c.c. After resection of

three fragments of muscular tissue from the infundibular obstruction,[30] a supraventricular tachycardia developed followed by shock and finally death 3 hours postoperatively. At autopsy, a laceration in the right coronary cusp of the aortic valve was present, in addition to cerebral edema, infarction of the liver, and arterial and venous thrombi in the lung.

7. *Mitral and Tricuspid Commissurotomy* (1 death).—This death occurred in a 34-year-old woman, operated upon through a right thoracotomy. Both valves were indurated and calcified, and, although both were satisfactorily opened, two to three plus mitral and one plus tricuspid regurgitation was produced. There was also an associated mild aortic stenosis. The patient developed hypotension at the end of the operation and died 4 hours postoperatively in spite of supportive measures. Postmortem examination of the heart specimen on the Presbyterian pulse duplicator[31] showed the mitral and tricuspid valves to be inoperable.

8. *General Considerations.*—Among 53 cases of rheumatic cardiac disease, there was a possibility of rheumatic activity in 12.

Seven of 67 patients received no digitalis prior to admission but 6 of these were digitalized during the hospital course. Of the 66 patients receiving digitalis or one of the purified derivatives, 27, or 41 per cent, showed evidence suggestive of digitalis toxicity.

Pericardial symphysis was present in 11 instances (16 per cent), 1 of these being incomplete. Of these, 3 instances were associated with previous cardiac operations although 1 was in the patient with constrictive pericarditis.

At operation, 31 (46 per cent) of the 67 patients showed unequivocal and 6 showed possible increased myocardial irritability. Twenty-five patients, or 37 per cent of all the deaths, showed ventricular fibrillation or cardiac arrest during surgery. Of these, 7 were resuscitated but died in the immediate postoperative period.

Among the 25 operating room cardiac arrests or instances of ventricular fibrillation, 10 were associated with unequivocal evidence of sudden or severe blood loss. The median blood replacement in these 10 cases was 2,500 c.c., with a range of 1,000 plus to 5,000 c.c. Despite the fact that considerable care was taken to replace blood loss, these patients tolerated even small deficits very poorly. A definite diminution in blood pressure (either abrupt or gradual) occurred in 19 (76 per cent) from 4 to 30 minutes preceding cardiac asystole. The blood pressure fall started with inception of anesthesia in 1, with placement in the lateral position in 1, on opening the pericardium in 5, with cardiac instrumentation or manipulation in 8, and with sudden blood loss in 2. There was no hypotension in 6 and, in 2 in which the aorta was cross-clamped, there was hypertension. Cardiac asystole occurred in the intrathoracic part of the operation in 15 instances, and after completion of that portion of the operation in 10. Eleven had evidence suggestive of digitalis toxicity and all 25 showed myocardial irritability. Intravenous procaine or procaine amide was used as

an emergency measure in 14 instances. In 12 of the 25 patients, measurement of left ventricular end diastolic pressure was performed and showed a median of 20 mm. Hg and a range of 12 to 44 mm. Hg.

It is interesting that of 22 patients who died in the first 48 hours after surgery, 20 (91 per cent) showed shock as part of the death picture, and 6 (27 per cent) showed frank congestive heart failure. (The shock and congestive heart failure were primary entities following surgery and not merely terminal events.)

It is also interesting that all 4 immediate postoperative deaths following mitral commissurotomy were associated with cerebral emboli.

Of 27 patients who died later than 48 hours after operation, 15 showed shock in the immediate postoperative period and 24 (89 per cent) showed frank congestive failure.

CONCLUSIONS

The data suggest that cardiac asystole, the cause of the operating room deaths, is associated with sudden or severe blood loss, definite periods of pre-arrest hypotension, increased myocardial irritability, and increased left ventricular end-diastolic pressure. The immediate postoperative deaths seem predominately related to post-surgical shock and to a lesser extent, frank congestive cardiac failure. The delayed postoperative deaths are almost invariably related to the presence of frank congestive failure. In most cases the shock and the congestive failure (not merely terminal events) were related to one or a combination of the following: (a) failure of the myocardium, despite good mechanical correction of the lesion, (b) aggravation of a pre-existing lesion, (c) production of a new lesion, and (d) failure to correct the mechanical lesion.

The exact place, in the above scheme, of other factors, such as digitalis toxicity, rheumatic activity, pericarditis, and myocardial dilatation or hypertrophy is limited by our lack of knowledge pertinent to these phenomena. Far-advanced tissue damage and anatomic derangements of the valves themselves, of the myocardium and of the pulmonary vasculature, are, at present, major insurmountable obstacles in the practice of cardiac surgery.

SUMMARY

A review of 67 consecutive closed cardiac surgical deaths, occurring over a 5-year period, is presented.

Major groups consisting of patients with acquired valvular heart disease are compared.

Some general correlations relating the types of cases, types of operation, the time intervals between significant events in the clinical course, various factors appearing to have a role in the death of the patient, considerations of a technical nature, and all available objective data of a physiologic or of a pathologic character have been attempted.

Some general conclusions are suggested.

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DISCUSSION

DR. GABRIEL P. SELEY (New York, N. Y.).—I would like to mention a complication that we have experienced in cardiac surgery, that is, gastrointestinal bleeding, especially in children. This work is from the service of Dr. Baronofsky, Mount Sinai Hospital, and was reported in the March-April issue of the *Mount Sinai Journal* by Drs. Kreel and Zaroff.

The first case was that of a 13-year-old boy who had a huge patent ductus successfully divided and sutured. On the fifth postoperative day he developed massive hematemesis and melena. He required 2,500 c.c. of blood. Nine hours later he bled again, required another 2,000 c.c. of blood, and about half an hour later developed evidence of massive perforation into the abdominal cavity and died.

Postmortem showed an acute perforated gastrointestinal ulceration with bleeding and perforation as the cause of death. The patent ductus was successfully closed.

In addition to this, there was a 14-year-old boy who had an interatrial septal defect closed with cardiopulmonary bypass. On the twenty-first day postoperatively he developed gastrointestinal bleeding and required 2,500 c.c. of blood over a period of 7 days, and finally recovered. X-rays showed a healed duodenal ulcer.

The next case was that of an 11-year-old boy with congenital aortic stenosis successfully repaired with cardiopulmonary bypass, who, on the second postoperative day, had massive tarry stools, required 1,000 c.c. of blood, and recovered.

The fourth case was that of a 17-year-old girl who had an interatrial septol defect successfully closed, who, on the fourth postoperative day, developed gastrointestinal bleeding not requiring transfusion.

The fifth case was that of a 37-year-old man who had a right atrial tumor removed successfully with the use of a pump. On the seventh postoperative day, he had 2,000 c.c. of gastrointestinal bleeding and he finally recovered.

We mention these cases because one should be careful to observe the patients carefully postoperatively, transfuse them if necessary and, if absolutely indicated, operate to stop the hemorrhage.

DR. JULIO C. DAVILA (Philadelphia, Pa.)—It is our feeling, likewise, that there is very little to discuss in this sort of presentation.

In undertaking this review, it was hoped that some overlooked factors, something perhaps that we have been missing in our day-to-day experience, might have turned up. As it is, our findings are in very general agreement with our day-to-day experience, and I am sure with that of most of us. They confirm that the lessons that we learn on each case are generally valid observations.

Dr. Seley's remarks about gastrointestinal bleeding are of interest. We have not seen a significant incidence of this complication. I recall at the moment only 2 cases in our entire experience, 1 operated at a hospital other than that from which these cases are derived. This patient had a resection of a left ventricular aneurysm performed by Dr. R. P. Glover and died on the tenth postoperative day of peptic bleeding.

The other one, a little girl who was at Presbyterian Hospital after having surgery for mitral insufficiency and after having a hemorrhagic gastrointestinal problem postoperatively, recovered without further complications.

The word this year is, "It's the pathology that counts." This is the lesson that we have all learned in the past 10 years of cardiac surgery. We must continue our efforts to identify and realistically appraise this pathology and so operate on those for whom we can accomplish something, and save some patients, for whom we can obviously do very little, from the trials and tribulations of surgery.

THE USE OF STEROIDS FOR THE CONTROL AND PREVENTION OF SERIOUS RESPIRATORY EMBARRASSMENT DURING AND AFTER INTRATHORACIC OPERATIONS

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AN INCREASING number of patients with greatly diminished lung function are seeking surgical treatment for a variety of intrathoracic lesions. The respiratory impairment may be apparent before surgery; it is frequently due to bronchial asthma, emphysema, or a combination of both. In other patients demonstrating good pulmonary function before surgery, accidents sometimes happen, such as aspiration pneumonitis and atelectasis, that may seriously embarrass respiration. In still others, an unusual form of respiratory distress may occur that is characterized by intractable bronchospasm, usually precipitated by periods of hypoxia and hypercapnia. This report is concerned with our experience with the use of various steroid compounds to control and prevent respiratory embarrassment in 26 such patients subjected to a variety of intrathoracic operations.

MATERIALS

The patients were divided into four categories. In the first category (Table I) were patients with asthma that was nearly always associated with moderately severe to severe emphysema. These patients were prepared for surgery with the steroids given in amounts ample to relieve all asthmatic wheezes, if these were present, even though large doses had to be given over several days' time. In the postoperative period the steroids were continued for 2 to 7 weeks. One patient in this group had had bleeding from reflux esophagitis associated with hiatus hernia, as well as a history of a duodenal ulcer. No complications occurred in this individual. Five patients were submitted to surgery because of large space-consuming emphysematous cysts. There were no persistent air leaks or difficulties with wound healing. Two of the patients had maximum breathing capacities below 30 per cent of normal. A tracheotomy was done in all 5 of the patients with emphysematous cysts. The following case history is cited as an example of the patients placed in this category.

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This investigation was supported in part by Research Grant H-1782 C4, National Institutes of Health; and the Duke University Regional Center for the Study of Aging.

Received for publication July 27, 1959.

TABLE I. GROUP I: CONTROL OF ASTHMATIC ATTACKS WITH STEROIDS IN PATIENTS SUBMITTED TO A THORACOTOMY

NO.	SEX	AGE	DIAGNOSIS	OPERATION	COMMENT
1	M	41	Hiatus hernia; repeated hematemesis	Repair	Asthma controlled
2	F	60	Granuloma of lung	Excision of subsegment of lung	Asthma controlled
3	M	50	Middle lobe bronchiectasis	Middle lobectomy	Asthma controlled
4	F	10	Middle lobe bronchiectasis	Middle lobectomy	Asthma controlled
5	M	50	Severe emphysema; lung cyst	Resection of cysts	Preoperative MBC = 28% of normal; no complications
6	M	78	Carcinoma of lung	Anterior segmental resection of upper lobe	Asthma controlled
7	M	67	Severe emphysema; lung cyst	Resection of cyst	Asthma marked; controlled, MBC = 64.7% of normal
8	M	58	Recurrent pneumothoraces	Excision of apical cyst	Asthma controlled; MBC = 27.6% of normal
9	M	54	Lung cyst; emphysema	Resection of cyst	Asthma controlled; preop. MBC = 56% of normal
10	M	64	Mediastinal lymphosarcoma	Anterior thoracotomy and biopsy	Asthma controlled

CASE I.—The patient, a 67-year-old man, had suffered from bronchial asthma for 10 years. In the last year the attacks of asthma were associated with alarming episodes of cyanosis and respiratory distress requiring many hospitalizations for the administration of oxygen. X-ray studies revealed a large emphysematous cyst in the right upper lung field which compressed more than 50 per cent of the right lung. There was also a smaller cyst on the left. On admission, the patient's maximum breathing capacity was 64 per cent of normal. The residual capacity to the total capacity ratio was 46.3. Helium clearance was 6 minutes. This patient was prepared for excision of the cyst in the right lung with 40 mg. of prednisone daily. During the operation and for the first 3 postoperative days, he was given 200 mg. of cortisone acetate intramuscularly, and then shifted to prednisone 40 mg. daily. At operation, a large emphysematous cyst was removed. A tracheotomy was performed in the operating room as a further safety measure and allowed to remain in place until the sixth postoperative day. Since this patient was a severe asthmatic, he was discharged with instructions to take 20 mg. of prednisone, 2 per day for 4 weeks. He was then to gradually taper off the drug dosage for a period of 2 weeks. The patient's postoperative course was entirely uneventful.

In the second category (Table II), the patients had marked emphysema, but they did not give a history of asthma or other evidence of allergy. One patient with a hiatus hernia was 85 years of age, while 2 others were over 75 years of age. Steroids were begun 24 hours before surgery and discontinued after 5 to 21 days. In none of the group were there any cardiovascular, pulmonary, or gastrointestinal complications following surgery. A patient's history is outlined below to illustrate the method of handling this group of patients.

TABLE II. GROUP II: PATIENTS TREATED WITH STEROIDS BEFORE AND AFTER SURGERY BECAUSE OF EMPHYSEMA AND ADVANCED AGE

NO.	SEX	AGE	DIAGNOSIS	OPERATION	COMMENT
1	M	80	Carcinoma of esophagus	Exploration of mediastinum	No postop. complications
2	M	75	Carcinoma of lung	Superior segmental resection	No postop. complications
3	F	85	Hiatus hernia; hematemesis	Repair of hernia	No postop. complications
4	M	51	Emphysema; carcinoma of lung	Biopsy of carcinoma of lung	No postop. complications
5	M	46	Moderate emphysema; carcinoma of lung	Left pneumonectomy	No postop. complications; preop. MBC = 57% normal; no aeration of left lung
6	M	64	Emphysema; carcinoma of lung	Biopsy of carcinoma of lung	No postop. complications

CASE 2.—The patient, a woman, was 85 years of age and had had symptoms of indigestion, regurgitation of stomach content, and heartburn for 10 years. Two years before admission she fractured her hip and had been bedridden since that time. Because this condition required almost constant bed rest, her symptoms were greatly aggravated. In addition to heartburn, she had acute episodes that were characterized by severe epigastric pain, nausea, and vomiting of coffee-ground material. Morphine and constant aspiration of the stomach with an indwelling nasogastric tube were necessary for relief. In spite of the patient's age, it became necessary to repair the large hiatus hernia. The patient was then given cortisone acetate, 100 mg. intramuscularly twice daily, for 3 days before surgery. Using general anesthesia, the hiatus hernia was repaired through a left thoracotomy incision. Following operation, the patient was continued on 200 mg. of cortisone acetate daily for 3 days. She was then given prednisone, 10 mg. 4 times a day for 5 days. After this, the steroid was gradually reduced until finally, after 10 days, it was discontinued altogether. This patient's postoperative course was not complicated by either respiratory, cardiovascular, or gastrointestinal symptoms.

In the third group of 6 patients (Table III), the indication for the steroids was to control the infection that followed the spread of secretions into the nonoperated lung that occurred during anesthesia. In 3, this was due to partial collapse of the operated lung and the squeezing out of secretions into the tracheobronchial tree with inadequate suction during the course of the operation. In 3, the spread was due to tuberculosis, while in 1 it was from coccidioidomycosis. The most striking physical sign was profound respiratory embarrassment accompanied by many expiratory wheezes. With the intravenous administration of 100 to 400 mg. of hydrocortisone, the relief was dramatic in all instances. The patient with coccidioidomycosis eventually died of disseminated disease. There was 1 patient of the 3 with tuberculosis who died, but this was not due to a dissemination of the disease but to coronary thrombosis. The following case history is an example of the patients in this category.

CASE 3.—The patient, a man, was 63 years of age and had a suppurative lung lesion of months' duration due to an unknown pyogenic organism. This had been treated vigorously all this time with antibiotics, but there still remained a large thick-walled cavity.

TABLE III. GROUP III: PATIENTS WHO RECEIVED STEROIDS IN POSTOPERATIVE PERIOD FOR ACUTE DISSEMINATED BRONCHOPNEUMONIA

NO.	SEX	AGE	DIAGNOSIS	OPERATION	COMMENT
1	M	71	Coccidioidomycosis	Exploration for carcinoma of lung	Died 3 weeks postop. of disseminated infection
2	M	56	Pulmonary tuberculosis	Excision of superior segment	Died 10 days postop. from coronary occlusion
3	M	46	Pulmonary tuberculosis	Segmental resection	Recovery
4	F	60	Pulmonary tuberculosis	Segmental resection	Recovery
5	M	63	Pyogenic abscess	Right middle and upper lobectomy	Recovery
6	M	50	Pulmonary tuberculosis	Upper lobectomy	Recovery

The question of carcinoma was not completely resolved, so excision was undertaken. At operation, a large abscess involving the right upper and middle lobe was removed. It was noted immediately after operation that the patient was in marked respiratory distress and that he did not improve in spite of frequent aspirations and positive pressure oxygen. He was noted to show extreme expiratory effort and loud expiratory wheezes were audible. A tracheotomy was then performed, but this resulted in only slight improvement. Because of continued respiratory distress and cyanosis, he was given an injection of 200 mg. of hydrocortisone intravenously and then given 200 mg. more in an intravenous drip over the next 12 hours. This resulted in immediate and dramatic improvement in the patient's respiratory symptoms. All of the expiratory wheezes disappeared, and the cyanosis showed gradual improvement over the next 2 hours. After the first 12 hours, the patient was carried on intramuscular cortisone acetate, 100 mg. twice daily, and, after the seventh day, this was gradually withdrawn until he was off steroids entirely at the end of 2 weeks. Erythromycin, 1 Gm., and 1,600,000 units of penicillin were given each day. The patient made an uneventful recovery in spite of the fact that x-ray studies showed diffuse bronchopneumonia throughout the unoperated side. This condition eventually cleared completely, and there was no residual infection or disease in either the operated or unoperated lung.

In the fourth group (Table IV) were patients who developed severe bronchospasm either during surgery or in the early postoperative period. These patients denied ever having had asthma. In 2 instances, the onset of the expiratory obstruction occurred more than 48 hours after surgery and was associated with proved respiratory acidosis. In both instances the bronchospasm could not be corrected with aminophyllin and epinephrine, but it did respond to large doses of intravenous steroids. As soon as the bronchospasm was corrected, the blood gas and pH studies returned to more normal levels, and the other signs of respiratory acidosis, such as clouded sensorium, unconsciousness, and arterial hypotension, disappeared. In one instance, the initiating factor was inadequate ventilation which prevented the unoperated lung from being adequately aerated. The bronchospasm developed during the time of surgery. In spite of the fact that the position of the endotracheal tube was corrected and the chest was closed, the persistent bronchospasm was not relieved until 400 mg. of hydrocortisone was administered intravenously. In another instance the patient had a large intrathoracic tumor that inter-

fered with ventilation. This was followed by bronchospasm that was corrected by the intravenous administration of 200 mg. of hydrocortisone. One patient, who had cirrhosis of the liver, eventually succumbed from liver failure. The following summary is an example of the employment of cortisone in this group of patients.

TABLE IV. GROUP IV: PATIENTS TREATED WITH STEROIDS IN POSTOPERATIVE PERIOD OR DURING OPERATION BECAUSE OF ACUTE RESPIRATORY EMBARRASSMENT ASSOCIATED WITH INTRACTABLE BRONCHOSPASM

NO.	SEX	AGE	DIAGNOSIS	OPERATION	COMMENT
1	M	42	Esophageal varices; cirrhosis of liver	Ligation of esophageal varices	Respiratory acidosis 8 days after operation; died 28 days later of liver failure
2	M	49	Hiatus hernia, recurrent	Attempted repair; malplaced endotracheal tube	Intense bronchospasm only relieved by 400 mg. of I.V. cortisone; recovery
3	M	58	Carcinoma of lung	Pneumonectomy	Respiratory acidosis; recovery
4	M	62	Carcinoma of lung	Biopsy of carcinoma of lung	Bronchospasm, relieved in O.R. by 200 mg. I.V. cortisone

CASE 4.—The patient was a 43-year-old white man admitted to the hospital with a history of repeated gastrointestinal hemorrhages that had begun suddenly 3 weeks before. Finally after 1 week of observation in his local hospital, the abdomen was explored for a possible bleeding peptic ulcer. At this time the stomach and duodenum were normal, but the patient was found to have marked cirrhosis of the liver. A balloon tampon was passed into the esophagus and stomach, and the hemorrhage was then well controlled. After 2 days, it was removed; but the hemorrhage recurred, and for this reason the patient was transferred to Duke Hospital with the tampon still in place. After the abdominal operation, a tracheotomy had been done. Twelve hours after admission to Duke Hospital, a left thoracotomy was carried out with exploration of the esophagus and intra-esophageal ligation of esophageal varices. The response to surgery was satisfactory in spite of the fact that he had had a previous abdominal operation, was markedly overweight, and was intensely jaundiced. On the seventh day after operation, he was noted to be semicomatose but without evidence of recurrent bleeding and with no obvious deterioration in liver function. On examination of the chest, breath sounds were quite distant except for very faint expiratory wheezes. It was suspected that he had respiratory acidosis. Arterial carbon dioxide tension was found to be 101 mm. of mercury. The pH was 7.32 and oxygen saturation was 82 per cent. Artificial ventilation with a mechanical ventilator through a cuffed endotracheal tube was started and 200 mg. of hydrocortisone given in 1 dose intravenously with another 200 mg. allowed to drip in slowly. In the next 24 hours the patient's sensorium cleared markedly and breath sounds became more normal, although it was of interest that, as the ventilation became more effective, more expiratory wheezes could be heard. After 24 hours, ventilation was excellent and the respiratory wheezes had disappeared. The arterial pCO_2 had diminished to 77.2 mm. of mercury with a pH of 7.35 and an oxygen saturation of 100 per cent. Six days later the findings were further improved, the pCO_2 being 70 and the pH 7.39. Cortisone was continued for 10 more days, being tapered off in the latter part of this period until at the end of this time no more steroids were administered. The patient, however, succumbed 3 weeks later from progressive liver insufficiency.

METHOD OF ADMINISTRATION OF STEROIDS

Steroids were given according to several different plans. The patients given the drugs as a prophylactic measure received them 36 to 48 hours before surgery either as prednisone, 10 mg., or methyl prednisone, 8 mg., 4 times a day. The night before surgery and on the morning of surgery, they were given 100 mg. of cortisone acetate intramuscularly. This was continued at this same dosage twice daily until they were able again to take the steroids by mouth. As soon as the patients were ambulatory, the drug was discontinued by gradually reducing the dosage, beginning on the fifth to the fourteenth day.

In asthmatic patients, the steroids were given before operation until all expiratory wheezes disappeared. In the period after operation, this was continued from 14 to 42 days so that the impaired breathing function resulting from the operation would have had a chance to return to the preoperative levels.

In instances of acute respiratory distress, hydrocortisone or its equivalent was given by immediate intravenous injection, using a 100 mg. to 400 mg. dose. The control of bronchospasm was a good index of the effectiveness of the drug. In one instance, 400 mg. of hydrocortisone was needed at one time, followed by a continuous drip, so as to give 200 mg. in the following 24 hours.

DISCUSSION

The rationale for the use of the steroids in the patients outlined above is based upon several basic actions of the steroids. Perhaps the most important action is the one which prevents an intense inflammatory response to chemical and bacterial agents and to trauma.[1] There may be a considerable inflammatory response in the larger bronchi, trachea, and larynx to the instrumentation with endotracheal tubes and with administration of the anesthetic agents. These factors associated with surgical manipulation and compression of the lungs can in turn elicit the outpouring of mucus and the prevention of normal ciliary action in the bronchial mucosa. In the ordinary patient with a large respiratory reserve, this is not of great importance; moreover, it is quite easily controlled by adequate aspiration. But in the patient with low respiratory reserve, the increased secretions may so embarrass lung function that serious consequences can occur. The patients, who by accident get a disseminated pneumonitis on the unoperated side, may have barely enough reserve to survive the first few hours. When the local reaction becomes more intense with the passage of time, they may then reach the point that there is not enough alveolar surface remaining for survival. The inhibitory effect of the steroids on the inflammatory response in the bronchi and in the lung parenchyma may provide the functional margin needed for survival.

In some manner that is not clear, the steroids inhibit bronchospasm. The wide experience with asthmatics and with the 9 postoperative patients outlined here is ample proof of this fact.[2, 3] It has also been noted in animal studies that the steroids will relax the muscle in isolated bronchial segments.[4] In a previous communication,[5] we have postulated that respiratory acidosis

may result in bronchospasm, and our experience indicates that respiratory acidosis cannot be relieved until the bronchospasm is controlled.

The steroids also have a beneficial nonspecific effect that is difficult to pinpoint but does allow support to the debilitated patient. As a result of our favorable experience with the patients in this study who were debilitated, we have extended the use of steroids to more and more poor-risk patients. The sense of well-being, the lack of respiratory complications, and the control of fever have made this drug a most valuable adjunct to surgery of the chest in the aged and debilitated.

There are certain toxic effects of steroids that have to be considered, and it has been this fear, perhaps, that has prevented the more widespread use of these drugs in surgery. The principal complications come from prolonged usage. Administered over a few days' time, as in the nonasthmatics of our group, there is very little chance that there will be a significant reduction in the resistance to infection or a significant effect on wound healing.[6] It is also of great interest that where there has been acute tuberculous pneumonia, the steroids have served to tide the patient over for a few days and they have not, in our opinion, interfered with the control of the infection with appropriate antibiotics.

No gastrointestinal bleeding has occurred in any of the patients, in spite of the fact that many patients were emphysematous and some had a previous history of gastrointestinal bleeding. To reduce the possibility of peptic ulceration, the stomach was aspirated after operation, and this was continued until good gastrointestinal function returned.

There have been no significant electrolyte disturbances except in those patients who already had respiratory acidosis, and this has tended to be corrected as the respiratory acidosis has been relieved. Daily electrolyte studies have been done on most of these patients in the postoperative period.

Based on our experience, we now use steroids in the preoperative, operative, and postoperative periods in known asthmatics. In patients who have poor pulmonary reserve, the steroids are always used as a preventative. In elderly patients who, in addition to moderate emphysema, frequently have a moderate amount of debility, the help of steroids is used to carry them through operation.

Where marked bronchospasm develops during the course of the operation, such as we have noted in respiratory acidosis, intravenous steroids are given until the bronchospasm is corrected and good ventilation is obtained. In acute respiratory emergencies, where there is disseminated spotty atelectasis and bronchopneumonia, large doses of steroids are given intravenously along with appropriate antibiotics.

SUMMARY

The experience with 26 patients given steroids for either prevention or control of respiratory impairment after thoracotomy is reviewed. In none of the patients have complications occurred from the steroid therapy. In some,

with serious respiratory embarrassment, elective operations have been made possible by the prophylactic use of steroids. In others, who developed acute diffuse bronchopneumonia after operation or who developed respiratory acidosis with bronchospasm, it is believed that the steroids have been lifesaving.

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MALIGNANT TUMORS INVOLVING THE CHEST WALL

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AS THE GENERAL public has become increasingly aware of the importance of early diagnosis of cancer, an increasing number of patients with chest wall tumors have come to this clinic for treatment. During the period 1938 to 1958, a total of 339 patients were seen. In 302 of these cases, the masses represented benign growths, including nevi, lipomas, exostoses, and so forth, but in 36 instances the tumor was malignant. In one other case the initial histologic diagnosis was osteogenic sarcoma of rib, but after further biopsy study, the diagnosis of benign giant cell tumor of the bone was made. This case is included to indicate the problems inherent in the diagnosis of these tumors.

In addition to the usual growths of mesenchymal origin involving the somatic soft tissues of the thoracic wall, we have included melanomas and mesotheliomas which invaded soft tissue and a variety of bone tumors which, of necessity, had to be differentiated from soft tissue lesions by careful roentgen and biopsy study. Although the characteristics of such a conglomerate group of tumors vary considerably, certain clinical features are common to all, and this renders precise diagnosis impossible without careful roentgenologic and histologic study. Despite the varying cellular origins of these masses, treatment does not vary greatly; wide excision and selected radiation therapy are utilized in most cases.

As is common in surveys of this type,[1-4] the cell types varied so widely that the number of patients in the series with a tumor of one type was small, and over-all survival figures are thus meaningless. The various cell types encountered, together with follow-up results in each case, are presented in Fig. 1.

AGE AND SEX DISTRIBUTION

The median age of a patient at the time of initial diagnosis ranged from 40 to 49 years, with extremes of age from 14 to 69 years (Fig. 2). Only 2 patients were under the age of 20. The series included 25 men and 12 women. No connection between the development of thoracic tumors, and occupation or co-existing disease was apparent.

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Received for publication June 16, 1959.
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SYMPTOMS

The appearance of a painless mass on the thoracic wall was the most common reason for a patient to seek medical attention, being the cause of initial complaint in 20 of the 37 patients. Pain was apparently a later symptom, but was the initial complaint in 13 patients. Miscellaneous causes for seeking

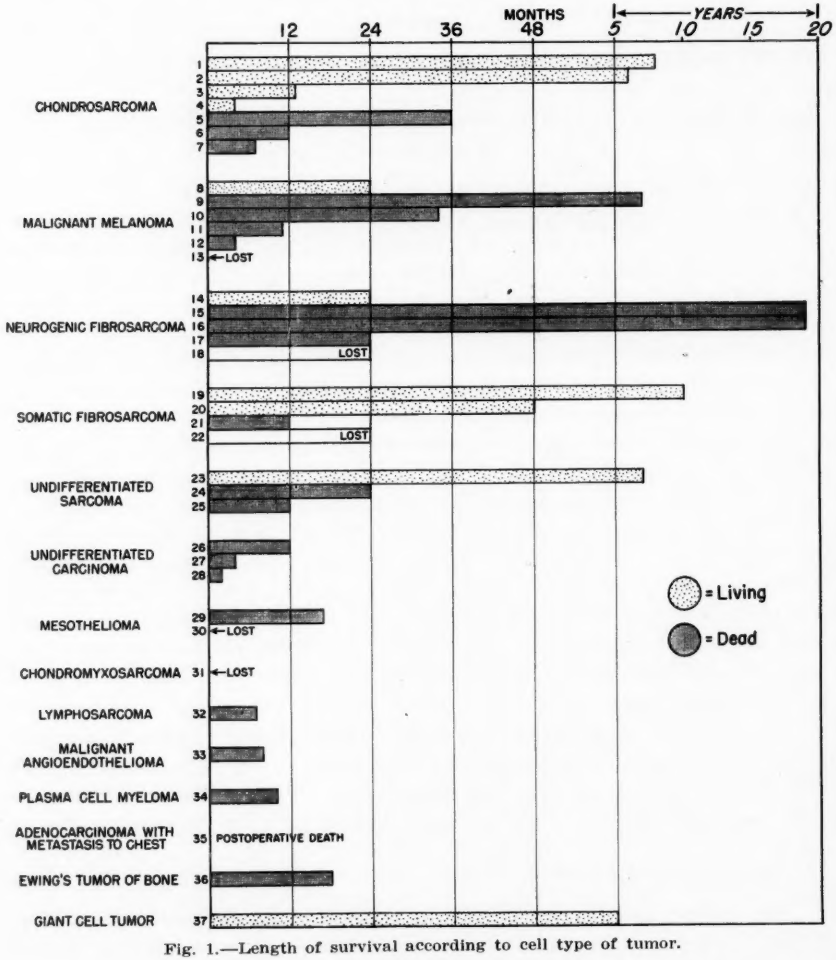


Fig. 1.—Length of survival according to cell type of tumor.

medical attention included shortness of breath in 2 patients with pleura encroachment, cough in 1 patient, and change of growth characteristics in a mole indicating the development of malignant melanoma in another instance. It would appear that, in general, the longer a symptom was present before operation, the longer that patient survived after treatment—suggesting that such

tumors grew slowly and were less likely to extend early beyond the confines suitable for adequate surgical or radiation therapy. In 3 cases, the development of a chest wall tumor was preceded by the development of an unrelated malignant lesion appearing years before the onset of the disease in the thoracic wall. A biologic tendency to the development of malignant disease may have been present in these cases.

DEFINITIVE DIAGNOSIS OF THE MASS

Since a painless lump was the most common presenting complaint, physical examination usually sufficed to establish the fact that a tumor was present, but histologic examination was essential before an effective therapeutic plan could be formulated. Whether histologic diagnosis was established by study of an incisional biopsy specimen or by block excision was governed by the size of the lesion, the clinical features indicative of the possible cell type, and the general

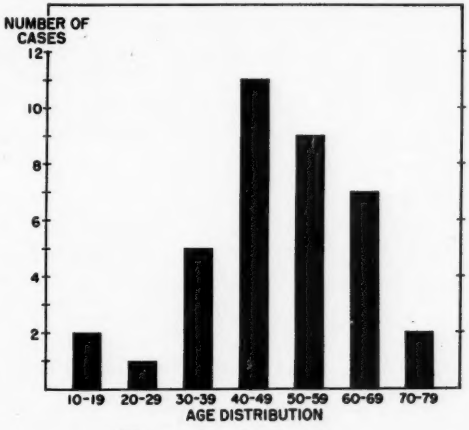


Fig. 2.—Age distribution.

physical condition of the patient. Since radical surgical excision is the most satisfactory therapeutic method in the majority of these cases, our efforts have been directed chiefly toward preparation of patients for initial radical excision; biopsy study has been reserved for the few questionable lesions of large size in refractorily debilitated patients with metastases or for those patients who might be expected to derive maximum benefit from an initial course of two million volt radiation therapy.

Before any surgical procedure is undertaken, be it biopsy or excision, a roentgenogram of the chest should be made. Further radiographic studies may be required to rule out the presence of a metastatic process.

Rapid frozen microsection is of limited value in these cases because of the subtleties of histologic diagnosis, and because bone and cartilage elements so frequently preclude this method of tissue sectioning. All biopsy incisions are closed meticulously. It is doubtful that a delay for paraffin microsection intro-

duces an appreciable hazard of metastasis, since any subsequent excision of a radical nature would encompass the entire area of biopsy. We have not used needle aspiration biopsy, preferring to have the greater bulk of tissue obtained by incisional biopsies available for study.

MICROSCOPIC CLASSIFICATION

In most of the groups, the definition of cell type follows a conventional type of classification. However, the fibrosarcomas have been divided into a somatic group, representing those tumors obviously arising from the fibrous components of muscle and connective tissue, and a neurogenic fibrosarcoma group, representing tumors arising from the stromal elements of nerves. The latter group has been classified by other pathologists as representing sarcomatous change within the neurilemmal sheath, and hence the term "neurilemmoma" has been applied to such lesions.

The undifferentiated sarcoma group either presented such an anaplastic picture that the origin of the characteristic cell type could not be determined, or represented highly malignant growths in which areas of different types of sarcomatous degeneration could be found (for example, anaplastic tumors containing elements of liposarcoma and fibrosarcoma). We believe that such undifferentiated sarcomas represent anaplastic lesions with multipotential growth characteristics.

MANAGEMENT

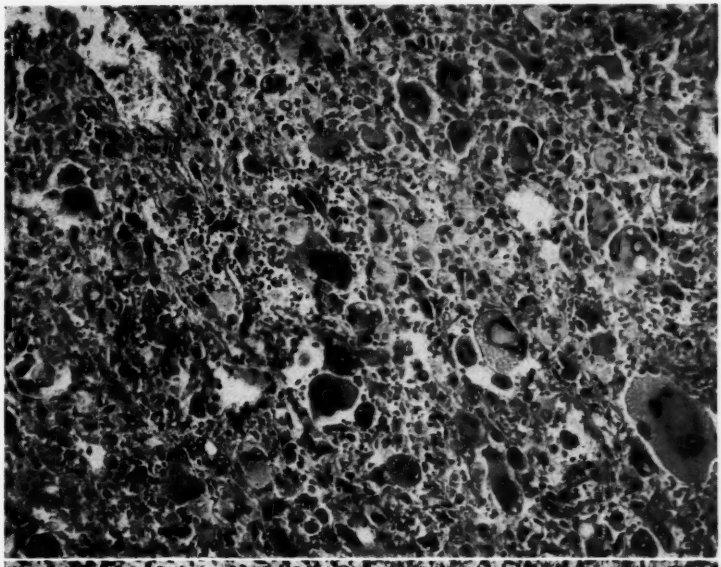
Every attempt has been made to obtain a wide block excision of the primary growth at the time of operation. Such excisions frequently required the removal of segments of ribs, costal cartilages, or sternum, either because of deep invasion of the more superficial tumors or because the tumor arose directly from these structures. In no instance was amputation of the shoulder girdle required.

Local recurrence was frequent in the various groups, particularly when the initial excision elsewhere had been patently inadequate. In a number of such cases, re-excision was possible and added appreciably to the survival time. Two patients in the fibrosarcoma group were submitted to re-excisions over a period of 2 to 4 years, and 1 of these is still living 2 years following a second excision for recurrence. A number of the block excisions involved the excision of multiple ribs and intercostal bundles. Prior to 1950, no attempt was made to apply struts or supports into the resulting extensive defect in these cases. Oddly enough, lung herniation did not appear to be a problem.

A small lump in a healthy, young patient would be considered favorable for complete eradication by excision biopsy with a generous margin of normal tissue, whereas large masses encroaching on thoracic viscera in a patient in poor physical condition would be sectioned for biopsy study during preparation of the patient for definitive excision, radiation therapy, or both.

The major error that arises in the management of these tumors, as in most mesenchymal tumors of the somatic soft parts, results from failure to make a

Fig. 3.



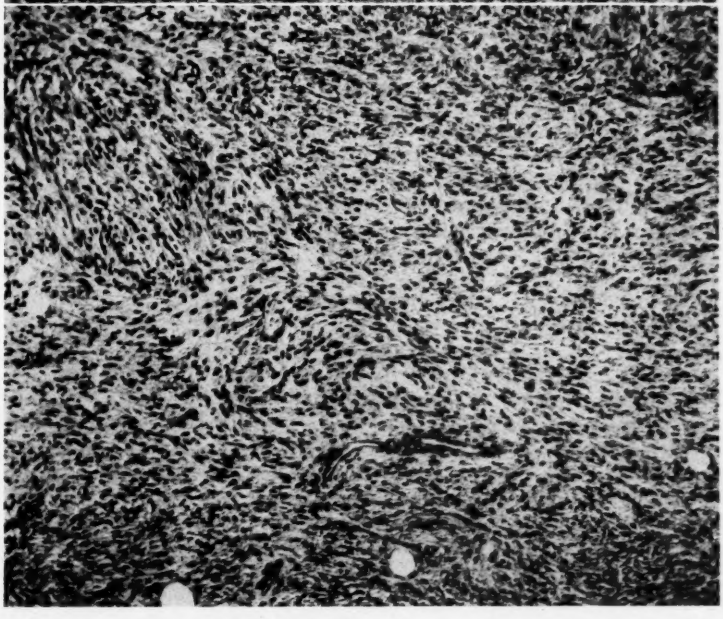


Fig. 4.

Fig. 3.—Undifferentiated sarcoma. Pleomorphism of cell elements and lack of characteristic architecture indicate the difficulty encountered in attempting to classify this type of tumor. (Hematoxylin and eosin; ×125; reduced ⅕.)

Fig. 4.—Fibrosarcoma, somatic type. Proliferating fibroblasts show irregular whorls without the palisade alignment characteristic of the neurogenic type. (Hematoxylin and eosin; ×125; reduced ⅕.)

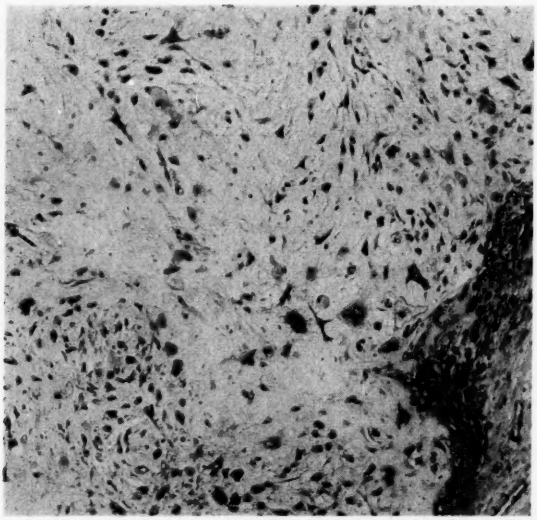


Fig. 5.—Chondrosarcoma. Characteristic distortion of cellular elements. (Hematoxylin and eosin; ×100; reduced ⅕.)

Fig. 6.—Neurogenic fibrosarcoma. This tumor is typified by the palisading arrangement of the fibroblasts which are thought to arise from the fibrous elements of nerve sheaths. (Hematoxylin and eosin; ×125; reduced ⅕.)

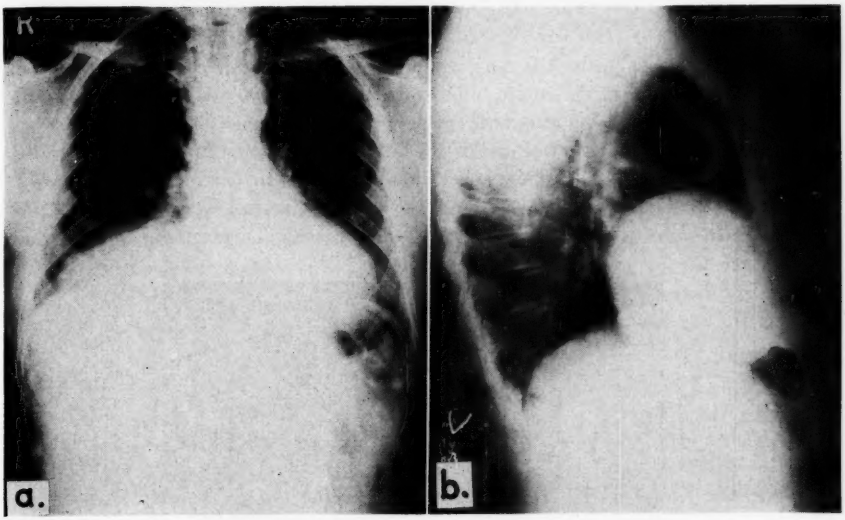


Fig. 7.—Posteroanterior (*a*) and right lateral (*b*) views in 62-year-old man, showing chondrosarcoma of costal cartilage. Mass was excised by block excision of ribs 6, 7, 8, and 9, lower sternum, and segment of diaphragm.

Fig. 8.—Lateral view, showing chondrosarcoma of sternum. Manubrium, upper two thirds of body of sternum, medial half of each clavicle, and anterior segments of ribs 1, 2, and 3 were later excised en bloc to remove mass.

sufficiently wide excision at the initial operation. The thoracic wall presents certain problems in the excision of complete muscle bundles, in view of the broad origin of the pectoralis major muscle anteriorly and resultant hesitation concerning excision of the entire muscle when this is involved by tumor. Moreover, the surgeon is reluctant to create large thoracic defects for fear of jeopardizing the patient's welfare in the immediate postoperative period by the creation of ventilatory insufficiency. This conservatism may result in unnecessary recurrence of tumor masses, and re-excisions in such areas are extremely difficult. It should be emphasized that wide primary excision is feasible and

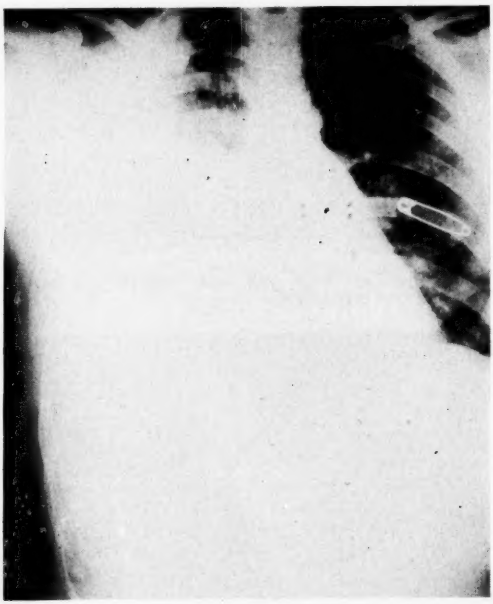


Fig. 9.—Posteroanterior roentgenogram made on sixth postoperative day, following excision of sternum, clavicle, and ribs as described in Fig. 8.

safe when applied to the thoracic wall. It should be possible to restore the integrity of the chest wall with suitable periosteal strips, autogenous bone grafts or temporary external supports but, even if this is not feasible, the body has a remarkable ability to adapt to extensive bony defects of the thoracic cage. This is strikingly illustrated in the following case report.

REPORT OF CASE

A 34-year-old mechanic first came to the clinic in 1951. He complained of a mass below the clavicle, which had been increasing in size for a period of 3 months before admission. Radical excision of this mass—a chondrosarcoma of the second costal cartilage—was carried out the same year. The manubrium of the sternum was removed in continuity with the upper two thirds of the body of the sternum, the medial half of each clavicle, and anterior segments of the first, second, and third ribs on each side. Severe

respiratory difficulty followed this operation as a result of paradoxical motion of the flail chest wall. This was treated only by a tight dressing and tracheobronchial catheter aspiration, although at the present time we would favor more vigorous therapy, such as external fixation and early tracheostomy. Approximately 500 c.c. of serous material was removed from the wound but the patient was well on the road to recovery by the twenty-second postoperative day. By the sixth week after operation the flail area of the thoracic wall was apparently stable. Sixteen months after operation, the patient was lost to follow-up. During the follow-up period, he showed no evidence of tumor recurrence, however.

The first information we had concerning the patient's ability to perform work following this extensive resection was obtained in a letter received 8 years after operation, in the course of preparation of this manuscript. Since it indicates better than any technical description the end result obtained without the use of struts, we have published the letter here in its entirety.

Dear Doctor:

In regard to your letter concerning my operation about six years ago, I had to be very careful about straining my chest. Standing upright I could do a normal day's work, but if I had to be under a truck and lift anything while on my back I would be useless. Standing to put my hands behind my head was uncomfortable and at times painful.

About a year or so ago, I was under a truck, it slipped off the jack and pinned me on the upper part of my chest within 6 inches of the floor. I lost about two weeks' work and then discovered as I regained my strength that I could put my hands behind my head, or lie on the floor and lift heavy objects without discomfort.

The last year I have been driving a motorcycle; it does not affect me in any way. My occupation is a truck mechanic—heavy equipment, and I now weigh about 180 pounds. The remaining effect from the operation, I believe, is poor circulation in the arms when I go to bed at night only; I toss and turn a lot during the night.

Although this type of physiotherapy (compression of the chest wall by a truck) is not recommended, it serves to indicate the ability of this patient to engage in active labor without discomfort or incapacity following radical excision of a malignant tumor in the thoracic wall.

RADIATION THERAPY

Since 1951, we have utilized two million volt x-ray therapy as an ancillary measure to wide block excision. The usual tissue dose is 7,500 r. In the absence of distant metastases, surgical excision was the primary form of treatment. Postoperative radiation therapy is reserved for those cases in which excision is incomplete because of extension into the vertebral bodies, spinal canal, heart, and other vital areas. In the presence of recurrences, a number of re-excisions might be attempted before radiation is employed. In the presence of distant metastases, surgical excision would be preferable to radiation therapy only if an emergency problem of ventilatory mechanics indicated that excision might possibly expedite disease control and prolong life.

TABLE I. SUMMARY OF CASES

CASE	AGE AND SEX	INITIAL COMPLAINT; PERIOD TO OPERATION	TYPE OF OPERATION	POSTOPERATIVE THERAPY	FOLLOW-UP REPORT
			Chondrosarcoma		
1	34 M	Lump 4 mo.	Block excision, manubrium, body of sternum, medial clavicles, ribs 1, 2, and 3	None	Living and well 8 yr.
2	31 F	Pain 36 mo.	Excisions of ribs 4 and 5 and spinal canal extension D_{11}-L_2	7,500 r 2 mev.	Living with disease 6 yr.
3	70 M	Lump 1 mo.	Block excision, ribs 7, 8, and 9	None	Living and well 1 yr.
4	62 M	Lump 4 mo.	Block excision, ribs 6 through 9, sternum, diaphragm		Living and well 4 mo.
5	47 M	Lump 24 mo.	Block excisions, multiple, of ribs 5 and 6, and 7, 8, and 9	7,500 r 2 mev.	Died of disease 3 yr.
6	49 M	Lump 12 mo.	Block excision, rib 9	7,500 r 2 mev.	Died of disease 1 yr.
7	56 F	Lump 6 mo.	Excision, left breast, chest wall, left lung tissue	None	Died of disease 7 mo.
			Malignant Melanoma		
8	47 M	Mole	Multiple local re-excisions 2 yr., soft tissues	None	Living and well 2 yr.
9	45 M	Lump 12 mo.	Multiple block excisions 5 yr., soft tissues	None	Died of disease 7 yr.
10	40 F	Lump 5 mo.	Block excisions; supraclavicular node biopsy 2 yr.	None	Died of disease 3 yr.
11	61 F	Mole	Excision, soft tissue		Died of disease 1 yr.
12	36 M	Pain 6 mo.	Block excision ribs 2 and 3		Died of disease 4 mo.
13	67 M	Lump 1 mo.	Block excisions, soft tissues	None	Recurrence in 2 mo.; lost to follow-up
			Neurogenic Fibrosarcoma		
14	58 M	Pain 1 yr.	Excision of mass, intercostal muscle	None	Well 2 yr.
15	48 M	Lump 6 yr.	Block excision of mass and intercostal muscle	None	Died 19 yr., without recurrent disease
16	68 F	Lump ?	Block excision, rib 9	None	Died 19 yr., without recurrent disease
17	57 M	Lump 4 mo.	Multiple re-excisions, pectoralis muscle	None	Died of disease 2 yr.
18	50 M	Lump	Multiple excisions, rib 6; ribs 4 through 9; excision of recurrence	None	Lost to follow-up 2 yr.
			Somatic Fibrosarcoma		
19	44 F	Pain 18 mo.	Block excision of ribs 8 and 9		Living and well 10 yr.
20	40 M	Lump ?	Block excision; re-excision at 2 years; ribs 1 through 4	7,500 r 2 mev.	Living and well 4 yr.
21	65 M	Pain 5 mo.	Biopsy of rib 6	7,500 r 2 mev.	Died of disease 1 yr.

TABLE I—CONT'D

CASE	AGE AND SEX	INITIAL COMPLAINT; PERIOD TO OPERATION	TYPE OF OPERATION	POSTOPERATIVE THERAPY	FOLLOW-UP REPORT
22	53 F	Lump ?	Block excision, ribs 4 and 5	None	Lost to follow-up 2 yr.
			Undifferentiated Sarcoma		
23	59 M	Lump 36 mo.	Block excision; 2 re-excisions (one year apart), soft tissues	7,500 r 2 mev.	Living and well 7 yr.
24	14 M	Pain 1 yr.	Excision of ribs and cartilage 3	1,200 r conventional to vertebral metastases	Died of disease 2 yr.
25	66 M	Lump	Excision, soft tissue	None	Died of disease 1 yr.
			Undifferentiated Carcinoma		
26	42 M	Pain 4 mo.	Biopsy, ribs showed undifferentiated carcinoma (from bronchus)	None	Died of disease 1 yr.
27	41 F	Lump 1 mo.	Block excision of sternum	None	Died of disease 4 mo.
28	69 M	Pain 6 mo.	Biopsy, rib 11; source unknown	None	Died of disease 2 mo.
			Mesothelioma		
29	46 M	Cough 1 mo.	Biopsy of pleura	Radiation	Died of disease 17 mo.
30	71 F	Pain 3 mo.	Biopsy of pleura	Referred for radiation elsewhere	Referred; lost to follow-up
			Chondromyxosarcoma		
31	30 F	Pain	Biopsy elsewhere; refused surgery	None	Lost to follow-up
			Lymphosarcoma		
32	52 F	Lump 3 wk.	Biopsy of rib 3	None	Died of disease 7 mo.
			Malignant Angioendothelioma		
33	16 M	Shortness of breath 6 mo.	Biopsy of pleura	2,400 r conventional radiation	Died of disease 8 mo.; necropsy showed disease
			Plasma Cell Myeloma		
34	59 M	Pain 1 mo.	Biopsy of rib 7	None	Died of generalized disease 10 mo.
			Metastatic Adenocarcinoma		
35	55 M	Pain 2 yr.	Block excision, lung and ribs 2, 3, and 4; adenocarcinoma of lung	None	Died postoperatively
			Ewing's Tumor of Bone		
36	39 M	Cough 1 yr.	Excision of ribs 8 and 9	Conventional radiation, nature unknown	Died of disease 18 mo.
			Giant Cell Tumor of Bone		
37	23 F	Pain 5 mo.	Biopsy of mass; excision of ribs 8 and 9 and mass, vertebral spine	1,990 r 2 mev. without response; then 2nd biopsy showed giant cell tumor	Living and well 5 yr.

SURVIVAL

Of the 37 patients in our series, 22 are dead, 10 are living, and 5 have been lost to follow-up (see Fig. 1). Of the 22 deaths, 20 were caused by the disease. Two patients lived 19 years after operation and died free of disease. In both of these cases, the original procedure was a block excision of a neurogenic fibrosarcoma, and further surgery was not required.

The 10 survivors are all well (the longest for 10 years) with one exception. The latter patient has metastatic disease from a chondrosarcoma and is failing. Two of the 5 patients lost to follow-up were known to be free of disease for 2 years. Although it might be reasonably safe to assume that they are still free of disease it is impossible to predict with any certainty what period of time can be considered "safe" in terms of cure; the underlying nature of the disease is the determining factor in this regard.

As we have stated previously the variety of cell types encountered and the number of cases involved in this series make analyses of survival of the over-all group relatively meaningless. Prediction of survival in any particular cell type is impossible.

SUMMARY

Between 1938 and 1958 a total of 339 patients with tumors of the thoracic wall have been seen at this clinic. In 303 of these patients the tumor was benign, but in 36 instances a malignant tumor was encountered. In addition to the sarcomas arising from the fibrous tissue or nerve elements of the thoracic wall, malignant melanomas and mesotheliomas presenting problems in management by involvement of deep structures of the thoracic wall were included, as well as a number of tumors of heterogeneous types which also presented problems in management because of their size or invasive tendencies.

Although the variety of cell types encountered in this group of 36 patients was so great as to preclude a statistically significant number of patients in any one category, it would appear that the chondrosarcomas and somatic fibrosarcomas were most amenable to treatment.

The initial presenting symptom was the appearance of a painless swelling on the thoracic wall; this was the cause for complaint in 20 of the 36 patients.

In most instances histologic diagnosis was established at the time of wide block excision of the mass. Incisional biopsy was reserved for the few questionable lesions of large size which were found in debilitated patients or patients with metastases.

At the present time, roentgen therapy, using a two million volt generator, is reserved for patients in whom distant metastasis is present, those in whom the disease was incompletely excised at the time of maximal radical surgery, extremely debilitated patients, and patients in whom a number of recurrences have been managed by re-excision.

In several instances, the local recurrence of tumors was managed by repeat excision, and these patients lived for several years following such procedures.

Patients with recurrent disease should not be abandoned but should be given every chance in view of the evidence, in a few instances here, that re-excision of recurrent tumors may result in a continuation of active life over a period of years.

Although the physician initially treating these lesions surgically may hesitate to excise a segment of thoracic wall wide enough to eradicate the tumor adequately, our experience indicates that conservatism in the extent of the resection is likely to lead to recurrence, and that such recurrent disease poses additional problems at the time of re-excision. At this time we are inclined to utilize the various techniques described for maintaining the integrity of the chest wall by the use of rib struts, periosteal strips, or external fixation. A number of patients early in the series who did not have such support, however, were able to acquire stability of the thoracic wall early, and went on to lead productive lives. To us, the error seems greater in the lack of adequate excision than in the creation of ventilatory insufficiency following wide excisions in the thoracic wall.

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SURGICAL TREATMENT OF AORTICOPULMONARY FISTULA SECONDARY TO AORTIC ARCH ANEURYSM

Report of Successful Case

James W. Giacobine, M.D., and Denton A. Cooley, M.D., Houston, Texas

ANEURYSM of the aortic arch is a serious disease which usually leads to death less than one year after the onset of symptoms.[6] In most instances the grave prognosis results from the tendency for aneurysms to encroach upon adjacent structures in the mediastinum frequently leading to rupture of the lesion with fatal hemorrhage. Perforation into the respiratory passages is apparently more frequent than rupture into vascular structures such as the vena caval system or pulmonary artery. In 1812, Wells[20] reported the first case of rupture of an arch aneurysm of syphilitic origin into the pulmonary artery producing an aorticopulmonary communication, and since that time a total of approximately 90 cases have been recorded. Thus, although the majority of syphilitic aneurysms occur in the ascending aorta where they are in intimate relationship with the pulmonary artery, it is interesting that rupture into the lesser circulation occurs in only 3 to 4 per cent of cases.[1]

Since Nicholson's[16] complete review of this subject in 1943, antemortem diagnosis of acquired aorticopulmonary fistula, secondary to aortic aneurysm, has become increasingly accurate as evidenced by the correct diagnosis in 8 of the last 10 reported cases.[2-4, 10, 12, 13, 15, 19] Onset of this complication is usually acute, with extreme dyspnea, cough, and precordial pain. Less commonly, the patient may remain relatively asymptomatic complaining only of mild dyspnea or precordial discomfort. The size of the fistula produced probably determines the severity of symptoms and clinical course. Although survival of 4 years has been reported in one case,[21] the general prognosis is poor, with survival averaging between 6 weeks and 4 months,[5] with death resulting from progressive right heart and subsequent left heart failure.[11, 17, 18]

After fistulization occurs, the anticipated signs of a shunt between the greater and lesser circulation occur. Pulse pressure widens and a water-hammer pulse becomes prominent. A thrill is often palpable over the pulmonic area of the precordium, and a continuous machinery-like murmur with systolic

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Supported in part by the C. J. Thibodeaux Foundation, Houston Heart Association, and the U.S. Public Health Service through Grant 3137.

Received for publication March 9, 1959.

accentuation is heard in the same location. Roentgenograms and fluoroscopy reveal the aneurysm with overactive pulsations in the pulmonary arteries. The electrocardiogram usually reveals right axis deviation and right ventricular hypertrophy, an interesting contrast to the left heart strain which occurs in the physiologically similar patent ductus arteriosus.

Among the explanations for this paradoxical finding is the concept that long-standing compression of the pulmonary artery, prior to rupture of the aneurysm, produces increased resistance to ventricular outflow which results in right-sided hypertrophy. While differential diagnoses include patent ductus arteriosus, congenital aorticopulmonary fistula, and rupture of the sinus of Valsalva, the most frequent incorrect diagnosis is thoracic aortic aneurysm with aortic insufficiency, in which the age group, etiology, physical signs, and x-ray findings may be very similar to those of the syndrome under consideration.

Although excision of thoracic aneurysms has become an accepted form of therapy during the past 8 years, repair of acquired aorticopulmonary fistula has not been undertaken until recently. In two recorded attempts at surgical correction, one patient survived exploration only and the other died of ventricular fibrillation during attempted removal.[10, 15] In both instances, the surgeon stated that the extensive inflammatory reaction between the aneurysm and pulmonary artery made repair of the fistula technically impossible. Recently, we successfully repaired an acquired aorticopulmonary fistula which we believe is the first successful case of its type.

CASE REPORT

J. P., a 45-year-old white man, was admitted to Methodist Hospital on Nov. 9, 1958, for treatment of an aortic aneurysm with suspected aortic insufficiency. Nine years previously hoarseness had appeared suddenly and he had received penicillin therapy for syphilis. Two years later he noted a sudden onset of weakness and paresthesia with swelling of his left arm. These symptoms abated somewhat after a week, but venous distention of the extremity persisted. Excessive activity resulted in recurrent swelling of this arm. During the 6 months prior to admission, a dull aching substernal and lower left chest pain was noted together with progressive dyspnea. Several months prior to admission he had developed a productive cough and chest pain and was treated unsuccessfully with antibiotics. He was referred for surgical evaluation with a diagnosis of syphilitic thoracic aortic aneurysm with aortic insufficiency.

Positive physical findings on examination included a blood pressure of 155/45 mm. Hg in the right arm and 160/40 mm. Hg in the left arm. Pupils reacted to light and on accommodation. A tracheal tug was present. Prominent distention of the superficial veins of the left arm, shoulder, and left pectoral region was evident but was not accompanied by venous pulse or increased skin temperature. The heart was enlarged to the left. A continuous murmur with systolic accentuation localized in the left second and third interspaces was noted, and peripheral pulses were water hammer in type.

Usual laboratory studies were within normal limits except for a positive serologic test for syphilis. Venous pressures as measured in the upper extremities were 14 cm. of water bilaterally. Circulation time, arm-to-tongue, using Decholin was 16 seconds. The electrocardiogram revealed right axis deviation and minimal right ventricular hypertrophy.

Roentgenograms of the chest revealed a mass in the superior mediastinum which was considered to be an aneurysm of the aortic arch (Fig. 1). An angiocardiogram confirmed the presence of the arch aneurysm and strongly supported the clinical diagnosis

of aorticopulmonary artery fistula since reopacification of the pulmonary artery occurred after the contrast material had entered the aorta (Fig. 2).

Operation was performed on Nov. 11, 1958. A left anterior third interspace incision was carried across the sternum for a short distance into the right third interspace. Approximately 350 c.c. of serous fluid was found in the left hemithorax. A large saccular aneurysm, measuring 12 cm. in diameter, arose from the posterior and left side of the ascending aorta, filling the aorticopulmonary window and compressing the origin of the left pulmonary artery (Fig. 3). Although the first portion of the descending aorta was aneurysmal on its entire circumference, the dilatation in the arch was confined to the lesser curvature. A continuous thrill was palpable in the left pulmonary artery, distal to an area of aneurysmal compression. The thrill could be obliterated by compression of the area of junction between the aneurysm and the pulmonary artery. In spite of the increased venous pattern on the chest and left shoulder, the innominate vein was not compressed by the aneurysm.

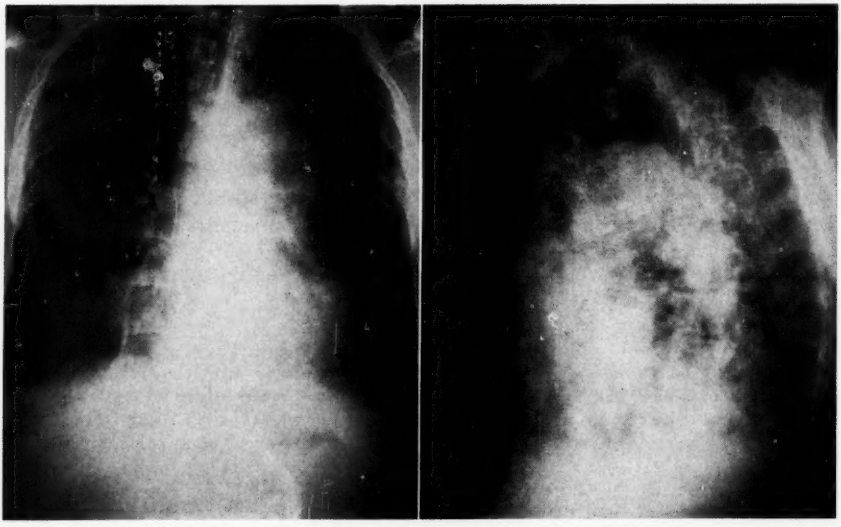

Fig. 1.—Roentgenogram of chest in 45-year-old patient showing mediastinal widening produced by an aneurysm of the transverse aortic arch encroaching upon the left pulmonary hilum.

Fig. 2.—Angioaortogram showing a fusiform aneurysm of aorta. Reopacification of the pulmonary artery is evident, suggesting the presence of an aorticopulmonary fistula.

The aorta and the left pulmonary artery were isolated at their origins by umbilical tapes. The main branches of the left pulmonary artery were isolated with heavy silk ligatures. The left subclavian artery was isolated and a tourniquet tape applied. The distal descending aorta was isolated with a tape (Fig. 3). The left phrenic nerve was preserved, but the left vagus was sacrificed since the recurrent laryngeal nerve had been compressed and destroyed by the aneurysm.

The left femoral artery was isolated and a catheter inserted for arterial perfusion. The pericardium was opened to expose the left auricular appendage, and a plastic catheter was inserted in preparation for left atrial to left femoral artery shunting using a single De Bakey roller pump (Fig. 3). The partial bypass was started using a flow of approximately 1,800 c.c. per minute, with the blood pressure in the right arm being monitored continuously.[8, 9]

A vascular clamp was applied to the transverse aortic arch with the blades placed somewhat obliquely across the base of the saccular aneurysm. The tourniquet about the left subclavian artery was tightened to prevent back bleeding, and the descending aorta was occluded distal to the aneurysm. The left pulmonary artery was occluded at its take-off from the main pulmonary artery with a vascular clamp, and branches of the left pulmonary artery were occluded with tourniquet ligatures (Fig. 3).

The left atrio-femoral bypass was begun at a rate sufficient to maintain a mildly hypertensive state in the head and upper extremities. The aorta was transected just distal to the proximal aortic clamp, and the wall of the saccular portion of the aneurysm was opened widely. Large amounts of laminated thrombus were removed. The fistulous communication between the sac and the pulmonary artery was readily visualized and found to be round and smooth in outline and to measure approximately 12 mm. in diameter.

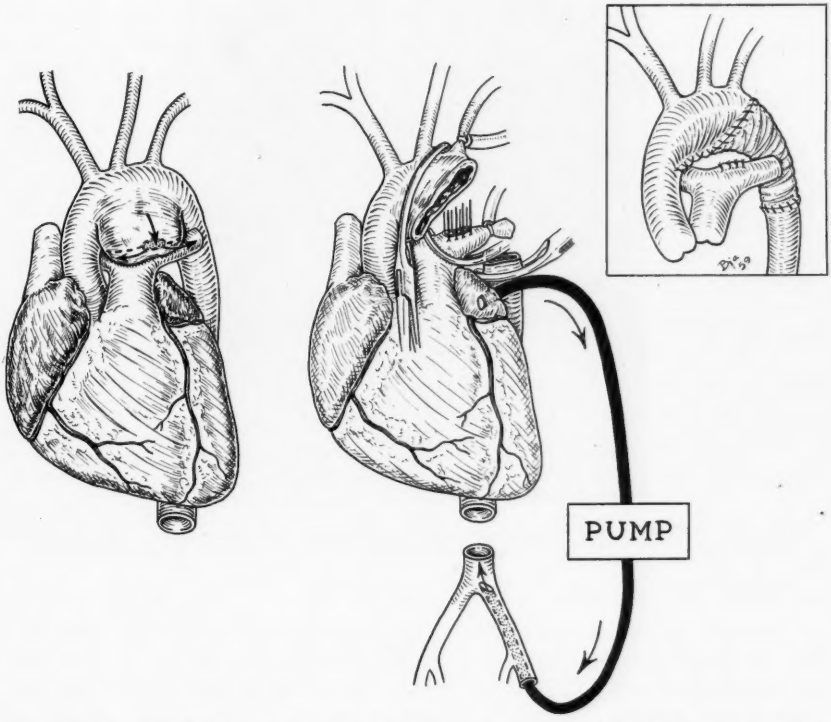

Fig. 3.—Drawing showing location of aneurysm of the aortic arch and fistulous communication with the left main pulmonary artery. Controlled extracorporeal circulation was used to bypass the aortic arch during resection of the aneurysm and graft replacement. Left main pulmonary artery was temporarily occluded, proximal and distal to the fistula. Inset shows completed repair using Dacron knit prosthesis.

The opening in the pulmonary artery was closed with interrupted sutures of silk. The remaining wall of the aneurysm was dissected from the mediastinum, and the descending aorta was transected proximal to the distal clamp. A lateral aortorrhaphy was completed where the origin of the aneurysm involved the ascending aorta. A knitted Dacron prosthesis, measuring approximately 12 cm. in length, was sutured in place between the arch at the level of the subclavian artery and the normal-appearing descending aorta, following which the clamps were removed and the partial bypass discontinued. The total time of aortic cross-clamping and of partial bypass was 55 minutes.

The postoperative course was uneventful. At the time of dismissal from the hospital 10 days later, examination revealed a normal precordium without murmurs or thrills. Aortic second sound could be heard distinctly. The blood pressure was 125/80 mm. Hg and the peripheral pulses were normal. Venous distention of the left arm disappeared. The patient was discharged on the tenth postoperative day in good condition. Examination 3 months later revealed no cardiac murmurs and a blood pressure of 140/80 mm. Hg. Roentgenograms of the chest showed a normal mediastinal configuration (Fig. 4).

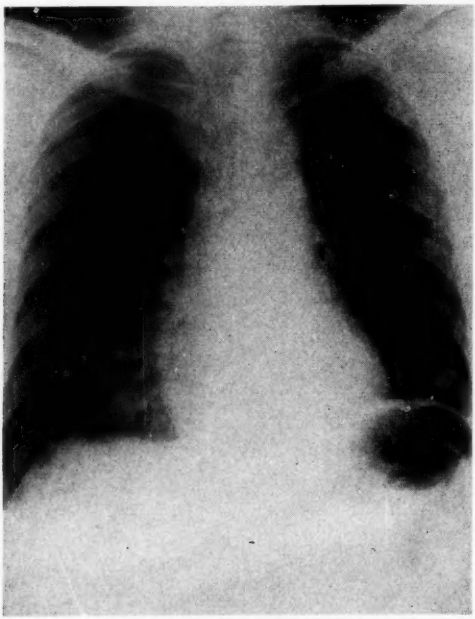

Fig. 4.—Roentgenogram of chest made 3 months after operation showing a relatively normal cardiac and mediastinal silhouette with slight elevation of left leaf of the diaphragm.

COMMENT

Rupture of an aneurysm of the aortic arch into the pulmonary artery produces an acquired aorticopulmonary fistula. This lesion is similar to congenital aorticopulmonary septal defect in that no duct exists which would permit safe interruption between clamps with closure by suture. Moreover, the presence of a dense inflammatory reaction between the pulmonary artery and aorta prevents separation of the two vessels by dissection. Thus, temporary diversion of the blood stream around the area of the fistula is necessary for successful repair.[7, 8] Temporary left atrium to femoral artery bypass, using controlled extracorporeal circulation, permits safe aortic occlusion at a point close to the heart. This technique controls left ventricular strain and prevents ischemic damage to the spinal cord during the period of aortic occlusion. Temporary interruption of flow through one main pulmonary artery as used in the reported case, or partial occlusion of the pulmonary artery with a curved exclusion clamp, isolates the

fistulous opening for repair. If the aorticopulmonary fistula involves the ascending aorta, temporary complete cardiopulmonary bypass with the pump-oxygenator should be used, similar to the method recommended for congenital aorticopulmonary defects.[7]

SUMMARY

Rupture of an aneurysm of the ascending aorta and arch is a rare complication usually leading to death within a few weeks or months. Surgical correction of the resulting lesion requires temporary diversion of the blood stream around the involved segments of aorta and pulmonary artery, excision of the aneurysm, and repair of the pulmonary arterial opening. In a 45-year-old patient, correction of an acquired aorticopulmonary fistula, secondary to aortic aneurysm, was successful. Excision of the aneurysm with restoration of aortic continuity by means of Dacron graft was performed, using a temporary extracorporeal pump-oxygenator bypass.

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